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In Memoriam

HAROLD EDMUND RYKERT,
M.B.(TOR.), F.R.C.P.(C.)

Dr. Rykert was born in Dundas, Ontario, in July 1904, the son of Dr. Arthur Rykert. He obtained his M.B. (University of Toronto) in 1928 and pursued postgraduate studies in England for a time. He was a member of the American College of Physicians and acquired his F.R.C.P.(C.) in 1943. He served with the R.C.A.M.C. overseas from 1939 to 1942, when, with the rank of major, persistent ill-health forced his return to Canada. At the time of his death, Dr. Rykert was Associate Professor of Medicine at the University of Toronto, and Senior Consultant in Cardiology in Sunnybrook Hospital.

As was stated so well in a recent motion* by the Faculty Council, "His friends always wondered at his never-failing energy which was directed by an intellectual appreciation of all his problems and a keen interest in everything that he undertook. . . . His interest was never limited to the field of cardiology. He was an excellent physician, competent in aspects of the care of his patients and in teaching students the basic principles of medicine and its practice. . . ."

On behalf of the student body, the JOURNAL can only add that Dr. Rykert's untimely passing was felt as a personal loss by all of us who had known him as a lecturer and clinician. We will always remember his great interest in us.

*Motion by Dr. R. F. Farquharson and Dr. R. Ian MacDonald, 30/11/54.

EDITORIALS

Not long ago a friend of ours was taken to the Emergeney Department of one of our teaehing hospitals for treatment of severe injuries and shoek that she had suffered in an automobile accident. A small group of keen young men wearing white eoats and "Dr. J. Doe" badges examined her and then withdrew several feet for a spirited diseussion of the ease. She overheard great differences of opinion expressed regarding diagnosis and treatment, and was seized by the very disturbing realization that her doctors were not equal to the situation. When one of them (older than the others) finally annouueed what was to be done, she received the decision with an uneasiness whieh persisted until someone told her that all the other "doctors" had been medieal undergraduates.

Thoughtless students can eause sick people considerable discomfort. Most of us serupulously avoid bedside expressions of horror or pity, or the use of such terms as "syphilis", "tubereulosis" and "eancer". But we may distress in subtler ways. In the ineident cited, mere disagreement among her "doctors" disturbed the patient far more than the taboo words would have done. Sometimes, rather than eonfessing that we are only students, we attempt sage answers to the patients' questions. We occasionally leave his (or her!) jacket untied, bed cranked down, bed-table out of reach, and radio turned off. Frequently, having finished with the patient, we linger at the bedside for prolonged discussion which could as well be eondueted elsewhere. The student who conscientiously warms his stethoscope-bell yet half-a-minute later eoughs in the patient's faee has created an uneoöperative subject. All these things are unkind, unnecessary, and poor public relations.

Usually these sick people are available for us to practise our arts upon because they are unable to pay for more privacy—let's not make needless discomfort their penalty.

W. H. S.

The New Year

To the final year, 1955 means graduation and interneship. (How far away it seemed baek in September 1949!) For the first premedical year, it is the next step toward distant 1960. To all the student body, the staff of the Journal sincerely hope that the new year will bring success and happiness in other pursuits as well as at examination time. We would also extend our good wishes for 1955 to the graduate readers and to our advertisers.

VERTIGO

I. G. L. VAN PRAAGH, 5T5

THE term "vertigo" which is derived from the Latin verb *vertere* meaning "to turn" implies, by derivation, a sense of rotation of the patient or his surroundings. Patients complaining of vertigo will describe a wide variety of sensations in addition to a sense of turning.

In an attempt to better understand and evaluate this confusing symptom, we shall define vertigo and discuss its anatomical and physiological basis, causes and treatment.

Vertigo may be defined as the consciousness of disordered orientation of the body in space and time (Brain). There are three ways in which the spatial orientation of the body may be felt to be altered:

1. The external world may appear to move in a rotatory fashion or in any other direction.
2. The body may be felt to be rotating, falling, or the movement may be referred to within the body, for example within the head.
3. The posture and movements of the limbs, especially the legs, are felt to be unsteady.

What determines whether the false sense of movement will be related to the external world or the patient himself is not known. Brain believes that it depends on the existing state of the proprioceptors of the body. Spalding feels that it is related to the personality of the individual—so that with extroverts the external world moves and with introverts the sense of movement is within the person.

Anatomical and Physiological Components

Several peripheral mechanisms with central connections give rise to afferent impulses that maintain spatial orientation. Vertigo is the disturbance of perception that develops when these mechanisms become imbalanced. These include afferent impulses from the retinae, skin and labyrinth, together with proprioceptive impulses from the ocular muscles, joints and muscles of the neck, lower limbs and trunk. The labyrinths are the most important peripheral mechanisms in the maintenance of spatial orientation, hence the severest and most clearly defined vertigo develops when their function is disturbed.

The membranous labyrinth filled with fluid endolymph lies inside the bony labyrinth, and is composed of three semicircular canals which lie at right angles to each other. One end of each canal is dilated into a fusiform swelling called the ampulla. The cavities of the canals open into an oval cavity in the bone, situated between the semicircular canals and the cochlea, known as the vestibule, where they are in close association with two membranous structures called the utricle and saccule which are known as otolith organs. The utricle communicates with the semicircular canals and the saccule communicates with the utricle and the cochlea. In the ampulla of each semicircular canal there is a branch of the vestibular nerve called the crista, which is surrounded by sensitive hair cells imbedded in a cap of gelatinous material called the cupula. The cristae are the proprioceptors of the canals, stimulated

by the flow of endolymph when the head moves in its plane. Movement of the head in any direction may excite labyrinthine impulses, but angular acceleration is the most effective stimulus. The utricle and saccule also receive fibres from the vestibular nerve, which enter at the maculae. When the head is erect, the macula of the utricle is horizontal, that of the saccule vertical. Fine hair-like processes of cells, acting as receptors, extend from each macula into a gelatinous substance on its surface in which small calcareous granules known as otoliths are embedded. The draw of these otoliths on the receptive hairs varies with the position of the head and therefore signals its position. While the semicircular canals react to movement, the otolithic organs are concerned in the orientation of the body with reference to gravity. The macula of the utricle is an organ of static sense, regulating the righting and stato-tonic reflexes, while the saccule is thought to have little or nothing to do with maintenance of posture, being mainly involved in cochlear function.

Labyrinthine impulses are conducted along the vestibular nerve, which arises from the vestibular ganglion in the internal auditory meatus. It passes through the cerebello-pontine angle as the medial part of the acoustic nerve, and on entering the brain stem separates from the cochlear division. Apart from some fibres passing directly to the cerebellum, the nerve terminates in the vestibular nuclei, which extend from the pons to the upper part of the medulla.

Secondary tracts are given off from the vestibular nuclei to the cortex, oculomotor nuclei, cerebellum and spinal cord. All the fibres to the cerebellum primarily enter the flocculonodular lobe which also sends fibres only to the vestibular nuclei (Larsell and Dow), and hence is concerned with the maintenance of balance.

The connections between the vestibular nuclei and the cortex are still not fully known, but current opinion suggests that there may be an area in the tempero-parietal region related to the reception of vestibular impulses.

Vertigo may be accompanied by incoordination of movement, nystagmus and other visceral symptoms due to the spread through the brain stem of impulses (largely of vestibular origin) such as pallor, nausea, vomiting, slow thready pulse, palpitations, sweating and sensations of heat and cold. Incoordination of movement is due to abnormal impulses from the vestibular nuclei passing to the skeletal muscles via the vestibulo-spinal tracts. Nystagmus accompanying vertigo is caused by abnormal vestibular impulses passing via the medial longitudinal bundle to the oculomotor nuclei. Of the two components of the nystagmus—fast and slow—the latter is more important, since it is a reflex response of vestibular origin resulting from abnormal stimulation of the semicircular canals. During an attack of labyrinthine vertigo the direction of the slow component of the nystagmus, together with the passpointing and falling, is toward the side of the lesion, while the quick component is directed away from the side of the lesion toward the more active labyrinth.

Clinical Considerations

To assess the clinical significance of vertigo, an aetiological classification will be used as a basis for discussion.

aural vertigo

The factors which may cause vertigo by producing imbalance between the two labyrinths are as follows:

Mechanical causes They include: wax in the external auditory meatus,

blockage of the Eustachian canal and sudden changes in atmospheric pressure.

Vascular Causes Very rarely a sudden attack of vertigo accompanied by a high degree of permanent deafness may be caused by a vascular accident such as hemorrhage within the labyrinth or thrombosis of the internal auditory artery.

Head Injury The labyrinth, which is very susceptible to damage, may sustain permanent injury following skull fracture or severe concussion. After the obvious effects of the injury have passed off, persistent dizziness and a disturbance of balance suggest the possibility of vestibular damage, even if there is no deafness. In some cases vertigo may be induced or accentuated by holding the head in certain positions, usually backwards and to the side.

Infectious Causes In labyrinthitis, secondary to otitis media, infection will spread inwards giving rise to vertigo, tinnitus and deafness, all of which may be continuous or recurrent. Occasionally vertigo may occur without either deafness or tinnitus. In chronic suppuration, the final erosion of the bony labyrinth may be preceded by slight dizziness which is increased by pressure on the ear—the so-called "fistula sign". This warning could signify the presence of a fistula between the internal and middle ear, and would call for early operative treatment. Difficulty may be experienced in differentiating between labyrinthine infection and a cerebellar extension. Vertigo, nystagmus and cerebellar signs may be present in both.

Nystagmus caused by acute suppurative labyrinthitis will be most active when the eyes are directed away from the side of the disease, the slow phase being toward the side of the lesion, and the fast component away from the side of the lesion, indicating that the function of the labyrinth on the side of the disease has ceased so that the healthy ear becomes the more active. In cerebellar nystagmus, the opposite is true: the slow phase is away from the side of the disease and the fast phase toward the lesion. A confusing differentiating point may arise in the initial stages of a serious labyrinthitis. Here the diseased ear is the more active one, hence the signs including the slow component of the nystagmus are to the opposite side. Rotatory nystagmus is more likely to be of labyrinthine than of cerebellar origin.

In Meningococcal meningitis infection may involve the labyrinth and be manifested as residual deafness and some disturbance in balance.

Mumps may be complicated by neurolabyrinthitis giving rise to acute labyrinthine failure. If bilateral it may cause total deafness.

Acute serous labyrinthitis, a spontaneous form of acute labyrinthitis, presents with rapidly progressive deafness associated with symptoms of acute vestibular disturbance: vertigo, nausea, vomiting, nystagmus, forced movements and ataxia of limbs on the affected side. These symptoms represent irritation of the affected labyrinth. The patient lies on his unaffected side and pass-points and falls to the side of the lesion. Nystagmus is rotatory and the quick phase is to the opposite side, to which the patient feels he is falling. Partial or complete recovery of both cochlear and vestibular function may occur.

In herpes zoster of the geniculate ganglion there are signs of acute vestibular failure, pain in the ear and mastoid region, radiating to the anterior pillar of the fauces and to the vertex, loss of taste in the anterior two-thirds of the tongue on the involved side, paralysis of the face on the same side if the infection spreads to the facial

nerve, and vesicular eruption of the ear drum, external auditory canal, pinna and palate on the affected side.

Noninfectious Causes *Spontaneous recurrent aural vertigo* was first postulated as being of labyrinthine origin by Prosper Méniere in 1861. The condition is characterized by severe attacks of vertigo, leading to vomiting and prostration and usually associated with deafness and tinnitus in one or both ears. It has been called *Meniere's Syndrome* or *Meniere's Disease*, the former term being used when the aetiology is unknown. The cases are believed to be due to a variety of causes, hence many conditions have been postulated as playing a part in the aetiology. These include abnormalities of fluid metabolism, sodium retention, local oedema due to local alterations in capillary permeability, vasomotor disturbance in the inner ear, focal sepsis, allergy, stenosis of the Eustachian tube, etc. Recent autopsy studies, beginning with the report of Hallpike and Cairns in 1938, have revealed a pathological condition common to a number of cases with Meniere's Syndrome. Dilatation of the endolymphatic system of the inner ear, in which the cochlear duct was dilated in all cases, the saccule and utricle in many cases, suggests the presence of increased endolymphatic pressure. This obstructive distension of the endolymph system has been called "idiopathic hydrops of the labyrinth". This must be distinguished from the hydrops resulting from serous labyrinthitis which shows a more widespread degenerative change (Lindsay).

Although we are aware of this condition's effect upon the labyrinth, the explanation of the signs and symptoms remain controversial. Hallpike and Cairns believe that attacks are due to a disturbance between the normal production and disposal of endolymph, causing a sudden rise in endolymphatic pressure, with "rapidly initiated bouts of asphyxia" of the vestibular organs.

The attacks of vertigo accompanied by deafness and tinnitus occur spontaneously, without any relation to activity in otherwise healthy adults, at intervals which vary from a few hours to several years. The first attack has an average age of onset of 49, men and women being affected in almost equal numbers. Usually the patient has suffered from a slowly progressive deafness and tinnitus in one or both ears for months or possibly years. Usually there is no warning before an attack but occasionally there is an increase in the intensity of the tinnitus and deafness before the onset. The attacks start abruptly, last a few minutes, rarely more than an hour, and terminate abruptly. During an attack the patient usually is prostrated, lying on the unaffected ear, and shows pallor, sweating and fine spontaneous lateral or rotatory nystagmus. Recovery is rapid, the vertigo subsides concomitantly with the nystagmus, and a feeling of unsteadiness on standing may persist for several hours.

The deafness in this disease is very variable, particularly in early stages. It may be more severe at the time of vertiginous attacks and may subside markedly between attacks. It is a perceptive deafness, showing either a uniform loss of hearing in the entire scale of the affected ear or a predominant loss of high tones. Distortion of loud, shrill, harsh tones is most painful to the patient. In many forms of deafness, the louder the sound, the better it is heard. Often in Meniere's Syndrome there is little difference in the loudness of a sound just being heard and one that is too loud for clarity and comfort. The deafness is caused by a distortion of the saccule and Reissner's membrane

associated with the hydrops with interferes with the sound transmission in the column of fluid in the cochlea.

The tinnitus which is usually worse at the time of the attacks of vertigo is described as a high pitched hissing, roaring or ringing, which fluctuates in its intensity. It is supposedly due to irritation of nerve endings, secondary to increased endolynph pressure, but since eighth nerve section does not usually abolish it, there is possibly a central causal factor in some cases.

It is important to realize that this condition shows a frequent tendency to spontaneous remission. In about 50% of long standing cases, attacks cease altogether for periods of months or years, although they eventually recur (Hyland). This must be seriously considered in evaluation of any method of therapy, since the institution of a definitive method of treatment and the occurence of a spontaneous remission may create a false causal relationship.

In most cases, attacks may be prevented or subdued by one form of *management* or another:

1. *Sedation* Use of barbiturates, antihistaminics, and the anti-motion sickness drugs such as Dramamine reduce the severity of attacks, but will not prevent them.

2. *Antiretentional regime.* Assuming that disturbed water metabolism plays a part in this illness, several varieties of treatment based on low salt intake and the use of ammonium and potassium chloride have been developed.

3. *Vasodilatation* The unproven theory that disturbance of the autonomic nervous control of the circulation of blood in the inner ear is a factor in the production of aural vertigo has led to the use of many agents including histamine, procaine, nicotinic acid etc. Histamine diacid phosphate seems to be beneficial in certain cases. Paralyzing the cervical sympathetic by injecting the stellate ganglion produces no sustained improvement.

4. *Destructive Procedures* Such treatment is necessary in unresponding unilateral cases. Deliberate intoxication of the labyrinth should be used only if there is bilateral involvment, since it will affect both labyrinths. Surgical procedures likewise are indicated in presence of intractable, crippling vertigo. The simplest and least harmful procedure should be used. Eighth nerve section, sparing the auditory fibres if possible, is the procedure of choice, especially if useful hearing is present on the side being operated on and if deafness has begun in the other ear. Destructive labyrinthectomy requiring exposure and removal of a piece of endolymphatic labyrinth is thought to combine the best results with the greatest safety. Tinnitus is usually decreased but hearing is destroyed. This method removes the dangers of intercranial surgery present in the operation of eighth nerve section.

Two other noninfectious conditions must be *differentiated* from Meniere's Syndrome;

Paroxysmal Positional Vertigo appears several seconds after the head assumes a certain position—usually backward and to the side. Vertigo and nystagmus rarely last more than ten seconds, can be fatigued easily by repetition, and will not recur for at least one half hour even if the offending position of the head is assumed again. The lesion is possibly in the utricle (Dix and Hallpike) or in the cerebellar-vestibular connections. Central causes resulting in paroxysmal positional vertigo may be differentiated by the fact that the vertigo and nystag-

mus are not fatiguable and the direction of the nystagmus may vary with position of the head.

Vestibular Neuronitis described by Dix and Hallpike is a sudden unilateral loss of vestibular function without any impairment of hearing. The lesion is thought to be somewhere in the vestibular neurones or in Scarpa's Ganglion. The acute vestibular failure is manifested by sudden onset of severe vertigo accompaniment by vomiting. The patient needs absolute bed rest, as the slightest movement of head or body may cause further vertigo. The symptoms slowly subside until after two or three weeks all is normal except a little residual dizziness on sudden movement of the head. This condition may be associated with a focus of infection, but often there is no causal factor. It may well be the condition known as "epidemic labyrinthitis".

Motion Sickness This is a disturbance of function leading to vomiting, vertigo and severe prostration, caused by repeated movement of the body in unaccustomed planes. It is thought that abnormal stimulation of the labyrinth is the most important cause. Visual, psychogenic and kinesthetic factors play a subsidiary role varying with the individual. Symptoms occur in the following order of frequency: vomiting, nausea only, headache, vertigo, abdominal discomfort, vomiting with nausea, lassitude, and blurred vision (Maitland).

Fixing the eye on a definite object in space is helpful in both air and seasickness. Nothing should be taken by mouth for the first few hours. Abdominal supports, by either reducing the movement of the viscera or by preventing venous pressure changes in the abdominal walls, have been helpful. Less disturbance is felt if the patient can lie supine. Many drugs have been advocated which either act as sedatives upon the whole body or effectively suppress the parasympathetic nervous system. The drugs most commonly used are Dramamine (which is a theophylline derivative of Benadryl), Hyoscine, and mixtures of Scopolamine and Benadryl.

Exogenous Toxic Materials Certain drugs will cause a toxic disturbance of the vestibular mechanism. Overdosage of quinine or salicylates may cause vertigo with a possible associated deafness and tinnitus, which suggests that the site of the toxic effect is the acoustic nerve, or the labyrinth, or both. Alcoholic intoxication and excessive tobacco inhalation may cause vertigo, possibly due to central toxic disturbance of the vestibular system.

Large doses and long administration of streptomycin will cause the development of deafness, low pitched tinnitus and vertigo. The newer dihydro compound has a greater tendency to cause deafness with less tendency towards tinnitus. If the drug is discontinued quickly at the onset of symptoms complete recovery is likely to occur. Even in the presence of total labyrinthine destruction, the patient usually recovers from the deafness and develops compensatory balance mechanisms so that there is no disability.

CENTRAL VERTIGO

Vertigo due to Lesions of the Brain Stem Lesions involving the vestibular nuclei will cause the same type of vertigo as seen in labyrinthine dysfunction, but it is very sensitive to movements of the head which may precipitate or aggravate it. Deafness and tinnitus characteristically associated with vertigo of labyrinthine origin are lacking. Headaches and vomiting preceding

the onset of vertigo suggest a central cause. The vertigo is usually non-episodic and commonly is accompanied by evidence of involvement of other cranial nerves, long tracts and cerebellar connections. Spontaneous nystagmus accompanying brain stem lesions may be horizontal, rotatory or vertical; the latter type, due to a lesion near the vestibular nuclei, is not seen in lesions solely involving the labyrinth. Positional nystagmus which is irregular in direction and changes direction with alteration in position is suggestive of a central lesion. This is in contrast to labyrinthine spontaneous nystagmus which is the same in all positions of the head.

Vascular accidents in the brain stem are most prone to cause vertigo. Thrombosis of the posterior inferior cerebellar artery causes severe vertigo at the onset, along with other characteristic findings. Although the vestibular nuclei are actually supplied by the anterior inferior cerebellar artery, severe occlusion of the posterior inferior cerebellar artery causes temporary interference with the circulation through the former vessel sufficient to temporarily disturb vestibular function by anoxia, to produce a very severe but rarely long-lasting vertigo.

A plaque of disseminated sclerosis in the pons will cause vertigo, which is likely to be transitory but may be severe and protracted accompanied by vomiting. Vertigo is occasionally seen in acute encephalitis and encephalomyelitis.

Vertigo is rarely seen in gradually developing lesions of the brain stem, such as tumours. Symonds suggests that when vertigo is noted in brain stem lesions it is probably caused by congestion or oedema of rapid development.

Vertigo due lesions of the Cerebellum Vertigo may be slight or absent in spite of massive lesions of the cerebellum, especially if limited to the lateral lobe. The majority of pathological conditions that involve the cerebellum also involve other structures, particularly the brain stem. This applies to vascular conditions such as thrombosis of the posterior inferior cerebellar artery, and to many inflammatory and degenerative conditions. If vertigo does occur with a lesion confined to the cerebellum, it is usually caused by involvement of the main cerebellar connections with the vestibular apparatus in the region of the flocculonodular lobe. Clinically, this is seen in children developing midline medulloblastomas involving the cerebellar nodulus, giving early disturbance of gait.

Vertigo due to Cortical Lesions Most common cerebral causes of vertigo are seen in epilepsy and migraine. A vertiginous aura in epilepsy may occur with seizures originating in the temporal and parietal lobes (Penfield and Foerster), and precede a convulsion or terminate in loss of consciousness. In migraine, vertigo is usually independent of other features. Shapiro suggests that paroxysmal vertigo, migraine and the aura of epilepsy are caused by vasospasm of some end-artery supplying the vestibular nucleus.

Raised intracranial pressure from any cause may produce a subjective sense of rotation when stooping. This is due to cerebral oedema and altered vascularity of the vestibular pathways to the cortex.

Tumours of the posterior cranial fossa, particularly if they involve the roof nuclei of the cerebellum, may cause postural vertigo and nystagmus. Tumours pressing on the vestibular nuclei

in the brain stem will likewise cause nystagmus and various degrees of dizziness. In subdural hematoma, vertigo is occasionally present, but is rarely a prominent symptom.

Vertigo due to Lesions of Eighth Nerve
Since the Eighth Nerve contains vestibular fibres, lesions of this nerve may cause giddiness associated with deafness and tinnitus. The usual site of involvement of this nerve is at the cerebello-pontine angle. Tumours of the acoustic nerve, particularly neurofibroma, are the commonest lesions, and though the vestibular nerve is nearly always involved with loss of peripheral vestibular function, vertigo is rarely prominent. This suggests that it is caused through involvement of the vestibular nuclei in the brain stem, either by the lesion directly, or through changes resulting from increased intracranial pressure. Cerebello-pontine angle tumours showing unilateral deafness and vertigo must be differentiated from Ménière's Disease in which vertigo occurs as an early sign. Usually on careful physical examination, a patient with a cerebello-pontine tumour will show evidence of involvement of the fifth or seventh cranial nerve or signs of cerebellar dysfunction, pointing to the side of the lesion.

Vertigo may result from compression of the nerve by aneurysms, abnormally distributed atherosclerotic arteries, or by involvement of the nerve in the inflammatory process of meningitis, arachnciditis or meningovascular syphilis.

When vertigo is associated with loss of consciousness, dyplopia, disturbances of sensation, failing vision, incoordination of movements or persistent spontaneous nystagmus, it is necessary to search for a cause within the central nervous system.

VERTIGO ASSOCIATED WITH CARDIOVASCULAR DISEASE

True vertigo is not common in cardiovascular and blood diseases, though transitory sensations of dizziness, unsteadiness, visual blurring related to change of posture or sudden exertion may occur. Most commonly the symptoms are allied to syncope rather than vertigo, and are likely caused by a general cortical anoxia. Dizziness may be complained of in postural hypotension, aortic valvular disease (particularly aortic stenosis), Stokes-Adams syndrome and severe cardiac arrhythmias. It is very uncommon in anemia, mitral valve disease and coronary thrombosis (Hyland).

Dizziness and occasionally vertigo may occur in cerebral vascular disease with or without hypertension. Symptoms are aggravated or initiated by change of posture or by exertion, suggesting that they are due to imperfect vasomotor adjustment as a result of diseased blood vessels.

Hypertension causing vertigo and dizziness is thought to be actually due to a recurrent local circulatory insufficiency in some part of the vestibular system, perhaps as a result of vasospasm. In cases where deafness is associated with hypertensive vascular disease it is thought that the internal auditory artery is one of the vessels involved.

PSYCHOGENIC VERTIGO

A sensation of instability with rotation, associated with anxiety and symptoms of sympathetic overactivity, is a common finding in neurotic patients. Brain suggests that it symbolizes the patients' fear of impending physical, mental and moral collapse.

Vertigo may be a conversion symptom in hysteria or be a complaint in schizo-

phrenic and depressed patients. It is very important to find other supporting evidence before concluding that vertigo in any patient is a product of a disordered mental state.

Summary

Vertigo has been defined and discussed with specific reference to its anatomical and physiological basis, clinical picture, causes (aural, central, cardiovascular, psychogenic) and differential diagnosis. This disturbing complaint must be evaluated in the presence of the total situation, which may be elucidated by a thorough history, meticulous physical examination, and appropriate tests.

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THE PHYSIOLOGICAL BASIS FOR HEADACHE

TREVOR OWEN, M.B., F.R.C.P.(Lon.) F.R.C.P.(C.),*

THE physiological basis of a sensation of pain may be a compound of elements due to stimulation of end-organs on the one hand and the appreciation of it in our consciousness on the other. We have to invoke what we must call a threshold of appreciation or perception in what Hughlings Jackson called, "the highest cerebral nervous arrangements then existing" and these are variable—the most unorganized cerebral activity. This threshold is at the point of the physiological process of mind beyond which we cannot go in physiological terms and the point at which we have to switch to psychological expressions, leaving physiology behind. In headache, as in pain elsewhere, we must remember these two elements, especially in chronic headaches. This is particularly true of the commonest of chronic headaches—the psychoneurotic, where the threshold for the appreciation of painful sensation may be so reduced that even normal sensory impulses may be perceived or interpreted as pain, without the presence of pain-producing substances or any other stimuli at all affecting end-organs. For in headache as such, the seriousness of the cause cannot be gauged by its severity.

May I remind you, briefly, of some *anatomical* points. The pain-sensitive

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structures of the head are all the extracranial tissues covering the cranium, especially the arteries (as we see in temporal arteritis), and of course the mucous membrane of the sinuses, and the periosteum. The localization of pain from these is reasonably accurate as contrasted with stimulation of end-organs inside the skull. Intracranial structures are not all pain-sensitive. Not the skull itself or its veins, not the brain tissue; most of the dura is not, most of the pia is not; the ependymal lining of the ventricles and the choroid plexus are not. The sensitive structures are the venous sinuses and their tributaries from the brain surface, parts of the dura towards the base and tentorium, the middle meningeal arteries, the cerebral arteries at the base of the brain, and the fifth, ninth, tenth cranial, and the first three cervical nerves. Intracranially then, as you approach the base of the brain and skull sensory nerves become more numerous—except superiorly, where there are the venous sinuses, their branches and the branches of the middle meningeal arteries. Pain from intracranial sources is referred pain like visceral or deep pain, and the sensory nerve supply is important. It is interesting and rather curious in some respects. The anterior and middle fossa structures —that is, as far back and including the superior surface of the tentorium—are supplied by the trigeminal, and the reference to the outside of the skull is roughly in front of the line drawn over the head between the ears. The posterior cranial fossa and the underside of the tentorium are supplied by the sensory fibres of the vagus and branches from the first three cervical nerves. You may well ask: why should the vagus be supplying sensory fibres to this area; do not these fibres arise in the jugular ganglion outside the skull? When one remembers the wide distribution and function of the rest of the vagus—respiratory, circulatory, and digestive—it is interesting to think about the biological reasons why information of what is happening in the posterior fossa of the skull should be available through this remarkable nerve—a great integrator of vegetative function. These sensory fibres of the vagus terminate in relation to the spinal tract of the trigeminal, which goes down as far as the level of the second spinal nerve. The vagus also supplies part of the back of the auricle and the posterior wall of the external auditory meatus; the trigeminal, the rest of it; and the glossopharyngeal, the tympanic cavity. The reason for this patchy state of affairs can probably be explained by a study of comparative anatomy and physiology; it now represents a vestige of a previous arrangement in animals where the ear and its auricle were bigger and more manoeuverable in some species, for hearing was a more important means of ensuring the safety of the animal than it is in the human today. However, we are still concerned with safety. Another curious fact is that the first three cervical nerves which share in the supply of the posterior fossa get there by travelling with the hypoglossal nerve. The best reason for this may be that the hypoglossal nerve is almost a spinal nerve itself, its nucleus being continuous, end to end, with the nucleus of the first cervical nerve.

* * *

I mention these things in this way not to add to the confusion when considering headache, but rather to emphasize the wide distribution possible in referred pain and in the disturbances from structures inside the skull and to arouse inquisitiveness into the reasons for it. Let us remind ourselves that symptoms are disturbed function, and that function and purpose determine structure—a fundamental principle of arrangements to which I conform, and of which we must

always be thinking. Changes in vascular structures and their tone play so large a part in the production of headaches that the nerve supply is important. It is not to be assumed of course that changes in tone are due solely to nerve impulses to the vessel wall.

* * *

Pain from intracranial sources, like visceral or deep pain, has faulty localization—except for the trigeminal and glossopharyngeal neuralgias which seem to be distinctive neurone pain. Intracranial pain is caused, apart of course from inflammation, by distortion and traction from displacement and distention of arteries and venous sinuses, and interference with cerebrospinal fluid circulation. Changes in C.S.F. pressure are often associated with headache. Yet increased pressure, as from tumours etc., does not always produce headache. Furthermore, lowering the pressure by drainage (as in lumbar puncture) will produce headache, presumably by making the brain sag on its moorings, which pulls on the venous sinuses and on the middle meningeal arteries and arteries at the base of the brain, making them distend, the cushion of fluid being removed. This type of headache is relieved by replacement of the fluid. However, raising the C.S.F. pressure to as high as 800 mm. or more of water will not cause headache. Thus it is probable that, for headache to be produced in tumours and other conditions which raise intracranial pressure in any way, there has to be distortion or an unevenness of pressure by twisting or partial blocking between parts, which creates a pull on the arteries at the base or on the basal dural attachments or venous sinuses. This headache is increased by jugular compression. In the headache induced by histamine and nitrites and in that occuring in fever, relief is obtained by increasing the C.S.F. pressure. This seems to indicate that such headaches are caused by distention of intracranial arteries (branches of the int. carotid), and that the application of increased internal support diminishes that distention and the amplitude of their pulsation. *This is not the case in migraine.* The vessels concerned in the headache of migraine are branches of the external carotid (which include the middle meningeal arteries from the internal maxillary)—particularly the ascending pharyngeal (which has branches to the middle and posterior fossae, as well as to the eustachian tube, orbital fat, eye muscles, and mucous membrane of the sphenoid sinus), the occipital artery (which supplies the trapezius and neck muscles), and the temporal arteries. I rather suspect too that the vertebral arteries may take part because, arising as they do from the subclavian arteries, they seem to belong to the extracranial family or group, as do the external carotid artery branches. The amplitude of the pulsations of the external carotid artery and its branches can be reduced by ergotamine tartrate if it is given early. The change in tone by itself may not cause pain, but the adventitia is sensitive to pain-producing substances and the long-continued dilatation produces thickening by oedema and the pain may last hours or days. When this point is reached ergotamine may not effect the calibre of vessels or the pain. This does not solve the difficulty of understanding the suddenness of the starting and stopping. The aura of migraine—that is, the visual disturbances, the paraesthesiae of the hand, lips and tongue (bilateral)—are most probably due to vaso-constriction causing local anoxia in the distribution of the branches of the internal carotid artery. Amyl nitrite will relieve these, but does not prevent the progression of the attack towards the headache which follows

after a short interval. This march of symptoms is reminiscent of epilepsy, and migraine in previous days was included with epilepsy in the group called the paroxysmal neuroses or "nerve storms", which seems a good name. This group included paroxysmal vertigo, somnambulism, and the night terrors of children. The headache component of migraine tends to obscure the full picture of the migraine phenomenon which shows itself in so much of the constitutional characteristics of an individual. The variety of attacks, in whatever system they happen, seem to be the result of an irregular accumulation of nerve cell irritability in the cortex or midbrain which explosively discharges. Other constitutional reactions are produced in this way. Similar, I think, is the so-called "histamine headache" (a poor name): a migraine-like unilateral headache of peculiar violence but short-lived (one-half to one and one-half hours), coming most often in the midst of the deepest sleep. In this there is lachrymation, scleral and conjunctival congestion, accompanied by dilatation and large amplitude of pulsation of the superficial temporal artery. This can be cut short by the injection of Novocaine around this artery. Excision of the artery does not prevent further attacks but may sidetrack them to another place. The artery is merely the end-organ, not the initiating focus. In my experience, the patient and his family are particularly free of migraine or its equivalents, and the attacks are widely separated in time so that they have a period of four to ten weeks every two to three years in which they have one or two every night. Histamine seems to have nothing to do with causing this phenomenon—it is unilateral, in the intervals histamine will not bring on an attack, and desensitization to histamine is unsatisfactory.

* * *

Psychoneurotic Headache is the commonest of all headaches and the mechanism is the same as in the headache of normal people. It is of two kinds, first the dilatation of vessels (probably branches of the external carotid) when it is throbbing, secondly from tension of the voluntary muscles of the skull and neck when it is a steady pain. The latter pain is the same as that due to "eye strain" from posturing in an attempt to counteract the difficulty. Also it often complicates the migraine headache. There is the other factor, that of threshold, where stimuli from the end-organs in the tissues mentioned above may be minimal or negligible; here the pain represents an awareness of structure, probably through positional sense. This is a symptom derived from the highest level, the physiological processes of mind, when the personality is disordered (delusional), or its relationship to the problems of life is unsatisfactory (i.e., the patient is worried about something). The head is identified as the core of the person, but the disturbance is physiological none the less.

* * *

The headache in *Essential Hypertension* is most commonly a disturbance of vascular tone or extracranial muscular tension differing in no way whatsoever from that occurring in the normal person, or in anxiety and psychoneurosis. It is not due to the pressure of the blood. Such patients may have been sensitive or "headachey" people all their lives, they may have migraine, or someone may have alarmed them about their blood pressure. The headache may sometimes be relieved by ergotamine tartrate, the blood pressure often rising at the same time. Where there is vascular degeneration in essential hypertension, vascular muscle fatigue may result in dilatation and pain; narrowing of the vasa vaso-

rum may lead to ischaemia of vessel walls, resulting in further damage and oedema of the adventitia. This causes pain; the next step, in crises of hypertension, is arteriolar necrosis in pain-sensitive structures. The many instances of diastolic pressures persistently at 140 to 150 without any headache seems to indicate that pain is due to the effects of severe damage in the vessel walls, not to the pressure of blood inside them.

When attempting to understand, in individual cases, the mechanism of the actual physiological basis for pain, anywhere, the differentiation between the possible causes is best arrived at by repeated experience in listening—unbiased and unhurried—to many patients' descriptions of pain in that region, at the same time thinking about and viewing with the widest vision the physiological purpose and the anatomical arrangements of the parts concerned and of the whole integrated person.

* * *

THE CARE OF THE UNCONSCIOUS PATIENT

H. J. M. BARNETT, M.D., F.R.C.P.(C.)*

THE unconscious state, if it is prolonged more than a few hours, is fraught with dangers. The hazards of the unconscious state are often as dangerous as the hazards of the disease process that had led to this loss of consciousness. Seldom in medical practice is it so important for the doctor to pay more rigid attention to simple but vitally important principles of general management as when caring for a patient who falls into this group. This care must be given from the very beginning and this requires that one recognize at once the conditions that are likely to lead to a prolonged period of unconsciousness. Cerebral vascular accidents, head injuries, intracranial infection, barbiturate and carbon monoxide poisoning would account for a large proportion of these cases. Accordingly, whenever one encounters a patient with one of them, the need for application of the principles to be discussed below should spring to prominence in the attending physician's mind. The following are of paramount importance:

1. Maintenance of an adequate airway.
2. Prevention of pulmonary collapse and infection.
3. Maintenance of hydration and nutrition.
4. Care of excretions.
5. Prevention of bed-sores.
6. Mouth care.

Looking at this list, the haughty student might consider that these are such simple principles of general nursing care as to be beneath his dignity. On the contrary, the wise student must recognize that a most important function of his handling of an unconscious patient is zealous supervision of the nursing care his patient is receiving. Good nurses as well as good doctors would subscribe to this generalization.

MAINTENANCE OF AN ADEQUATE AIRWAY

Brain tissues jeopardized by a vascular occlusion, concussed by the sheering forces of a closed head injury, inflamed by a pyogenic infection or damaged by a poison such as barbiturate in excess, is in no condition to tolerate any hypoxia that can possibly be avoided. Unfor-

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tunately, the unconscious state that often accompanies these conditions is of sufficient depth to reduce the cough reflex, to interfere with swallowing and because of flaccidity of oral and pharyngeal muscles to allow of an obstruction of the upper respiratory passages from the falling back of the tongue and the closing in of the flaccid pharyngeal muscles on the narrowed lumen. Struggling attempts to breathe more freely lead to further increase in mucosal secretions. The patient gasps through his mouth and dries out these secretions. Before long they are of sufficient quantity and tenacity to give a dramatic and serious tracheal or bronchial obstruction.

To avoid this, from the onset of the loss of consciousness one insists that:

1. The patient is kept off his back entirely. A prone position with the head turned to the side, or a lateral position, is essential. Turning every hour will prevent accumulation of secretions in the dependent passages.
2. No pillow should be used.
3. The foot of the bed must be raised at least eight inches.
4. If coma is deep and breathing stertorous despite these measures, a rubber or plastic airway to pull the tongue forward may be tolerated but must be regarded as potentially dangerous if not removed and cleaned often as it will increase secretions from irritation.
5. Conscientious but gentle use of oral and nasal suction through a soft rubber catheter attached to a vacuum bottle should be started at once before secretions dry and thicken. It is to be used every 15-20 minutes rather than waiting for signs of obstruction to appear.
6. Atropine is to be avoided. It reduces the quantity of secretions but those that are present become insipissated.

When does one advise a tracheotomy? If the airway is a prime concern of the attending physician *from the beginning* of therapy, tracheotomy will be necessary in only a small percentage of this group of patients. However, *as soon* as it is evident that the above measures have failed to prevent or remove evidences of respiratory difficulty, immediate consideration must be given to emergency bronchoscopy. This will generally need to be followed at once by tracheotomy. Once a tracheotomy is done, it cannot be assumed that the need for vigilance is past. Unless the inner tube is changed once or twice daily and, even more important, a small rubber catheter be used to apply suction directly through the tube, *every 10-15 minutes*, a complete block will surely ensure presently.

PREVENTION OF PULMONARY COLLAPSE AND INFECTION

To get rid of secretions is to get rid of the main cause of both atelectasis and secondary pneumonia. This problem has been covered under section I. The only additional suggestion is the use of daily prophylactic dosage of penicillin in patients deeply in coma and unlikely to recover in twenty-four hours.

HYDRATION AND NUTRITION

Hydration is vital. It has been shown in infants and young children that the oxygen content of the superior longitudinal sinus falls off precipitously when they become dehydrated. Presumably a lesser degree of this phenomenon is present in adults and it reflects hypoxia of tissues that cannot tolerate it.

Duodenal feeding is much more satisfactory than continuous intravenous therapy. Intravenous glucose is an inadequate diet and as the days stretch into weeks the additional possibilities arise of electrolyte upset and vitamin lack. Veins become inflamed and thrombosed. The hazards of continuing intravenous therapy in a comatose subject are greater

than the risk of carefully passing a duodenal tube.

CARE OF EXCRETIONS

Increasing restlessness in an unconscious person usually means one of two things—respiratory obstruction or bladder distention. Both are preventable.

If the patient has retention, a small Foley catheter with expertly supervised tidal drainage is recommended. If this expert attention is not available, a Duke's irrigation device is simpler to operate. If neither are used, twice daily irrigations must be used. If incontinence is the problem, as it usually is after the first few hours, a condom-catheter arrangement is preferred. The condom must not be too snugly attached or balanitis will ensue.

Loose stools are more common with duodenal feeding than is constipation. Should constipation occur, a small daily enema is a good method of bowel control. Paraffin oil is taboo because of the danger of lipoid pneumonia.

PREVENTION OF BED-SORES

Bed-sores begin either as superficial abrasions or as pressure-point sores. Bed-making done properly to prevent lumping and wrinkling, alcohol cleansing, powdering and strict control of urine and feces will keep the skin and subcutaneous tissues healthy. No amount of powdering and alcohol rubs, though, will prevent a deep bed-sore from starting as local muscle ischemia from pressure between a bony prominence and the mattress. The necrosis begins in the muscle while the overlying skin may still look healthy. To prevent this requires frequent turning.

Flanellette sheets absorb moisture better than do cotton sheets and are useful where long term nursing is going to be needed.

MOUTH CARE

This is not given for its aesthetic value. A dirty mouth will lead to serious infection such as Ludwig's angina or suppurative parotitis. Dehydration enhances the possibility. Thrice daily swabbing of the mouth with a weak antiseptic solution is essential. Dentures must be removed at once. The safest thing is to get into the habit of looking for them in all unconscious patients.

In Conclusion

No one can immediately predict the degree of recovery and ultimate future of a patient admitted to hospital in coma from a stroke or a head injury. Handel wrote "The Messiah" after he recovered from a cerebro-vascular accident. All should be treated vigorously even though one must admit that in a varying proportion of those who recover, a mental or emotional cripple may be the reward of one's efforts. Nevertheless, the doctor only need be pleasantly surprised once about the outcome to have it registered upon him that he is incapable of being the final judge of those to be regarded as beyond his help. In meningitis one can be equally and often more confident of the outcome than with strokes. Tuberculous meningitis affords nowadays an excellent example. Prolonged deep coma here is disturbing but is not incompatible with return to normality. In barbiturate poisoning the use of the principles outlined above is much more important than the use of stimulants, such as picrotoxin. It is not uncommon to see suppression of blood pressure and respirations respond dramatically to bronchoscopic aspiration where picrotoxin had originally seemed to be indicated. Picrotoxin would have been far less useful indeed as it is a convulsant. It would have been dangerous—even more than it is at the best of times.

PSYCHOLOGICAL MEDICINE IN GENERAL PRACTICE

V. J. BUTLER, 5T5

IN any discipline which professes to be scientific and unified, the tendency towards departmentalization must be offset by continual integration of the findings of the various areas of special study. In medical praetice an important sign of this integration, the increasing cooperation betwèen psychiatry and the rest of medicine, has been due in part to growing recognition of the futility of attempting to treat states of mind apart from states of body, or states of body apart from states of mind.

While it is almost universally recognized that psychological medicine is a basic art and science essential to the effective practice of sound elinieal medicine, few indeed are the medical schools which provide thcir graduates with adequate organized knowledge of its scope, importance, and practice.

This article will attempt to outline some elementary aspects of these questions.

The Scope and Importance of Effective Psychological Medicine in General Practice

Learning to handle patients with understanding, patience, and taet is a professional and economic necessity to every practitioner. Of special significance in this regard is his handling of the hostile patient: the physician who dismisses such a patient without attempting to discover and relieve the cause of the hostility (oftcn it is fear) performs a disservice to the patient, the medical profession, and himself.

Whatever branch of medieine he practices, every clinician should be well acquainted with the part played by prolonged cmotional disturbance in the production and modification of disease. For some time it has becn fashionable to apply the term "psychosomatic" to that group of organic diseases which seem to be in large part caused by, or at least frequently associated with, prolonged emotional abnormality: peptic ulcers, some forms of colitis, angina and many cases of hypertension, migraine, certain collagen diseases, and many skin and allergic disorders. However, while it has long been recognized that anxious or fearful patients do not recover as rapidly or as completely as they might otherwise, there is increasing evidence that their peace of mind is more or less indispensable to recovery: in every patient, by virtue of his illness, there are emotional problems which, if they remain untreated, may worsen the symptoms and the prognosis or add entirely functional symptoms. For this reason, Stafford-Clark suggests[1] that the term "psychosomatic" should signify a method of approach to the study of all disease which is based upon acceptance of the interdependence of mind and body and all it implies, rather than a particular group of diseases, and much less a particular specialty of medicine—it is in fact a method of approach which no good doctor ignores.

Functional illness, in which no eause but emotional disturbance can be demonstrated, accounts for 25-75% of the complaints dealt with in general practice. It will be considercd in more detail in a subsequent section; for the moment, it is sufficient to point out that it is fre-

quently misunderstood and mismanaged. While few people will not grant that such everyday phenomona as blushing and the symptoms of acute fright are definite physical changes reflecting a definite psychological state, it is often difficult for laymen, some students, and a few clinicians to understand how prolonged emotion may similarly be the cause of physical changes and symptoms in the functional disorders. Indeed, how often both the physician and his patient (who may have a patent emotional disturbance) are discouraged and disgusted when after numerous expensive investigations the disorder is labelled "functional". In such cases, disappointment is entirely unjustified, for the outlook for recovery in properly treated functional illness is better than it is in many truly organic diseases. In functional illness, unnecessary examinations and laboratory studies, coupled with placebo therapy (or, more commonly, its equivalent —drugs and other treatments when they are not indicated), fix the symptoms, assure the patient that informed medical opinion agrees with his own contention that he is suffering from an exotic organic disease, and make future treatment considerably more complicated and difficult.

When guesswork is used, there are many pitfalls in differentiating between functional disorders and both early organic disease and those primarily organic diseases which present most clearly as psychiatric disorders: islet cell adenoma, hyperthyroidism, hypothyroidism, some cases of Addison's and Cushing's diseases, confused or amnesic patients suffering from pernicious anaemia, congestive failure, or silent strokes, and toxicity due to drugs or bacteria. It is obvious that a history which is not organized to uncover psychological problems will be inadequate in such situations, for the practitioner cannot evaluate the organic and functional contributions to the patient's incapacity, and it is then all the easier to attribute functional symptoms to an organic lesion or, conversely, to attribute the early symptoms of organic disease to its functional accompaniment.

The clinician's diagnostic ability improves when he ceases to group the clinical features of emotional stress with the specific symptomatology of underlying organic diseases; his thinking on any particular diagnostic problem is clearer since his experience of various syndromes, being separate from the variety of functionally appended symptoms, is better crystallized.

There are many other aspects of medical practice in which effective psychological medicine is important: its application to *Obstetrics* by Grantly Dick Read and others has shown that much of the suffering which mothers have been indoctrinated by superstition to expect is needless and dependent upon fear (of pain, of the unknown, of death, and of the dependence of the child); other emotional disturbances to be guarded against include rejection of the child because of resentment at loss of beauty and freedom. Regarding *Surgery*, Ebaugh[2] draws attention to the acute anxiety reactions which may occur postoperatively, especially when the organ tampered with is "sexualized" (e.g., uterus, breast, prostate) or when the patient is a child under four submitted to a long operation under general anaesthesia. Tissue committees have found that surgeons are by no means exempt from mismanagement of functional illness in their own particular way. *Paediatric* psychology can obviate many later disturbances as well as dealing with behaviour problems, bed-wetting, and developmental retardation (which includes support and guidance of parents). In recent years, as the prac-

titioner has been armed with ever more potent and specific therapeutic agents for the battle against treatable diseases, he has tended to neglect incurable patients, giving less of his attention to providing them with the symptomatic and supportive measures that were the stock-in-trade of the physician of another era; as a result, they suffer more than ever from feelings of rejection and hopelessness.

The family doctor, who practices in the social context, is best equipped to deal with psychiatric problems as they arise, since he is more conversant than anyone else with his patients' stresses, their families, their principles of action, and their responses to previous stress. As his ability and confidence in the handling of psychological problems increase, he fairly exudes reassurance, his patients' well-being and total health improve, and he can do much towards managing very complex emotional situations. He is the first member of the psychiatric team, not only because he sees the patient first but also because he handles most cases. He must be able to screen psychiatric cases which present in his office, recognizing which ones he may attempt to treat alone, which ones he must refer, and which ones he can expect to be amenable to treatment (a wide range, today). His approach to referral is important; it is considered in a subsequent section on management (q.v.).

The management of acute psychiatric emergencies falls on the shoulders of the general practitioner, especially when his practice is at some distance from a large psychiatric centre. He must therefore be prepared to deal adequately with: acute alcoholic poisoning, chronic alcoholic addiction, drug addiction, threatened or attempted suicide (and recognition of potential suicide in the depressed state), the acutely disturbed psychotic, the confused elderly person, mentally defective children, and acute emotional disturbances in children (e.g., tantrums and breath-holding, acute anxiety and fear). It is not within the scope of this article to discuss the management of these emergencies.

Common Emotional Disorders

A CONSIDERATION OF CASES

When we survey the literature devoted to emotional disorders, we are at once struck by the lack of agreement on three important aspects: the meaning of terms in wide use, the causes and psychodynamics, and the clinical types encountered. The extent of this lack of agreement may be taken as an index of the need for further investigation and unification in this field. Seemingly dogmatic treatment of these topics in the following discussion should therefore be construed only as an attempt to combine consistency with brevity.

If we adapt the terminology of Watts and Wilbur[3] to that of our other sources, their views are capable of a broader application. They state in effect that a person who fails to make a healthy adjustment to emotional stress eventually develops functional symptoms (we shall see that there are alternatives) and, conversely, that functional disorders are invariably caused by emotional stress. The stress may be on a conscious or unconscious level, but in either case the patient may fail to appreciate the causal relationship between emotional stress and physical symptoms; he does not feel well and seeks medical attention, but only rarely (of his own volition) does he mention his emotional problems.

These authors proceed to classify emotional stresses as extrinsic and intrinsic: Extrinsic stress may occur (a) between psyche and soma (e.g., fear of real or imaginary disease), (b) between

psyche and social environment (e.g., religion, financial worry, dissatisfaction with work, marital and sexual problems, or disturbed relations with in-laws or parents), (c) between soma and social environment (e.g., post-traumatic fears of losing earning power). Intrinsic stress is neurosis; it is a deep psychological disturbance, difficult to treat because of its distant origin in poor emotional adjustment during infancy, childhood, or adolescence. Symptoms of extrinsic stress are aggravated when the patient finds himself in the stressful situation and subside when he is alone, while those of intrinsic stress are more pronounced when he is alone and unoccupied.

It is illuminating to view emotional stress within the framework of Selye's concept of the General Adaptation Syndrome. Selye demonstrated three stages of the organism's response to any stress, physical or psychic, moderate or severe in nature: (a) the alarm reaction, which is characterized by high-speed responses of the endocrine and autonomic systems, and resembles illness; (b) the stage of adaptation to the stress, an abnormal condition in which the organism is relatively impervious to the primary stress but abnormally susceptible to other stresses; (c) the stage of physiological exhaustion, which immediately precedes death when the stress is of fatal severity. When the adaptive stage fails, because the response is either inadequate or in itself incompatible with homeostasis, the symptoms of disease appear. The particular disease and symptom pattern produced by emotional stress depends on many factors (among them, the nature and severity of the stress, the patient's nervous and physical constitution, physical state, previous experiences, and present environment), but it will be one of three main types: functional illness (or functional symptoms added to those of underlying organic disease), neurosis (or psychosis), or one of Selye's "diseases of adaptation" (these coincide almost exactly with the so-called "psychosomatic" group which have already been described as seeming to be in large part caused by emotional stress).

CLINICAL FEATURES

Two pathways by which emotional stress may produce symptoms are described by Stafford-Clark[1]: The response may be discharged directly through the autonomic nervous system, so that the normal physiological accompaniment of the primary emotion is simply heightened or intensified; or, it may be discharged indirectly through conscious processes by which, through failure to deal with the conflict or accept the unbearably painful nature of the emotion, a kind of secondary tension is set up, and this may issue as a hysterical symptom, a symbolic act, a conscious and unpleasant feeling for which the patient can find no cause, or again as a disturbance of autonomic regulation (instead of being discharged through consciousness after traversing the higher pathways of the central nervous system).

If now we attempt to orient the functional disorders within a simplified classification of psychological disturbances—without considering the psychoses, mental deficiency, psychopathic personality, or the minor personality abnormalities seen every day (the temperamental and the selfish, eccentrics and most cranks, the infantile and the senile)—there are four more or less overlapping groups:

1. If there is no underlying psychological abnormality, emotional stress (e.g., working under tension, or worry associated with the incapacity of any illness) may exceed the patient's tolerance and produce a primary disturbance

of the autonomic regulation of any system: there may be palpitation and tachycardia with shortness of breath on exertion (the effort syndrome), anorexia and gastrointestinal disturbances, or mild disturbanees of temperature regulation.

2. In the presenee of a minor psychological problem, the symptoms of autonomic dysfunction produeed by stress of any kind become modified by extrinsic stress (e.g., fear or the patient's anxiety about the persistence of symptoms). This not only further disturbs autonomic function but also gives rise to distorted symptoms related to the patient's eoneept of his disease. Sueh functional symptoms are often superimposed on those of underlying organie disease; they often prove more disabling than the structural lesion.

3. Neurosis, a deep psychological disturbance usually resulting from failure to resolve stress in infaney, childhood, or adolescence, is itself an intrinsic stress which may be discharged either as a purely psyehie disturbance—hysteria (loss of a function or pain of unphysiological distribution), affective disorders (such as anxiety states or depression), phobias, obsessive-compulsive rituals, or symbolic symptoms—or as functional symptoms of autonomic imbalance.

Neurosis should be suspected when
(a) the symptoms are related to periods of emotional stress rather than to trauma or exposure to infection;
(b) the factors whieh aggravate and relieve the symptoms are suggestive of emotional disturbance;
(c) ancillary symptoms tend to round out a functional pattern rather than a recognizable organic syndrome;
(d) the patient's reaction to his symptoms is abnormal—undue apathy or, more commonly, undue concern—or he becomes upset while describing the situations whieh disturb him and angry when the doetor seems to be passing over the symptoms too lightly;
(e) the patient's complaint of exhaustion and weakness is out of all proportion to his appearance of ill health (Prof. Farquharson[4] remarks that this is almost pathognomonic of severe emotional disturbance of this type, even in the presence of serious organic disease).

However, the diagnosis of neurosis can be established only by investigation of the patient's psychological adaptation. These people look for some disadvantage which they suffer (such as illness) to aecount for their failure and their desire for help; they have never established principles of action for themselves, but overreact to fears and apprehensions, and become disabled by symptoms which are minor. When they discover any symptoms about which they may worry, a vicious circle is set up.

The past history is usually typical: During childhood the patient was considered physically and emotionally delieate (sensitive and easily brought to tears); phobias and nightmares plagued him and he may have wet the bed until 10 or 12 years of age. He was taken eut of school for long periods because of indefinable illness, suffered feelings of inferiority about his social shortcomings, and avoided competition (e.g., in sports) with his fellows. Adolescence brought new problems, perhaps a "nervous breakdown", and he developed specialized narrow interests to compensate for inferiority in other fields. As an adult he has needed mueh medical attention, and it will be noted that the treatments given could not possibly have been effeetive in the diagnoscd diseases. The functional enquiry will be most revealing: the patient has every eoneeivable symptom, the symptoms are vague and distorted, and they seldom

waken the patient from sleep even though they are excruciating. Subconscious slips may be revealing, as when one woman recalled and dated the onset of her symptoms by the coincident arrival of her father-in-law to live with her family.

4. The "diseases of adaptation" are probably caused by a combination of emotional stress, which exceeds the patient's tolerance for prolonged periods, and some physical sensitivity or constitutional inadequacy which channels the illness into a particular system. In every case, investigation should attempt to uncover any associated psychological problems, since their treatment may significantly alter the course of the disease. Further consideration of these diseases is beyond the projected scope of this article.

In the emotional disorders, the aim of examination should be the *total diagnosis* of all that afflicts the patient: this must include identification of symptom complexes resulting from both organic and functional causes, and an evaluation of the contribution of each to the total disability. In any patient, the diagnostic evaluation must include an estimate of the patient's ability to understand, resolve, or endure psychological stress.

THE MEANS AND GENERAL PLAN OF MANAGEMENT

If we remember that the basis of emotional disorders is the patient's unhealthy response to a stress which interrupts his striving towards a goal, it is apparent that there are four factors which we may attempt to modify in order to remedy the situation: the stress, the patient's response to this particular stress, his principles of action in the face of stress generally, and his goals.

The means of treatment may be classified as physical, psychic, and social: Physical treatment includes diet, exercise, physiotherapy, electro-convulsive therapy, and surgery (pre-frontal leucotomy), but *not* placebo therapy. Social therapy includes programming, group therapy, social casework, and occupational therapy. To laymen and most students the term psychotherapy (so often dismissed with one word in lectures and textbooks of medical management) signifies specific analysis, a means of treatment made unavailable to most practitioners by limitations of time and training. Actually, this limitation applies only to major psychotherapy, an attempt by an analytic specialist in psychiatry to change the basic attitudes and reactions of a patient.

Minor psychotherapy is, as Dr. Allan Walters aptly characterizes it, *the art of medicine*—every point of interpersonal contact between doctor and patient: the doctor's reputation, the attitudes of attendants (the patient's family or nurses), the intimacy of the doctor-patient relationship arising from transference and rapport, the patient's confidence and faith in his doctor, such hope, comfort, reassurance, and encouragement as can be transferred to the patient, suggestion (altering the stress felt by the patient), the able practitioner's intuitive knowledge of when to speak and explain and when to be a good listener, the insight the patient gains by telling his story to a sympathetic listener, counsel, guidance, and teaching, persuasion and exhortation or (if the patient cares for the doctor) reward and punishment, and management for recreation and refreshment with relief from stress. All these are available to every wise practitioner.

The importance of total diagnosis in emotional disorders has been mentioned; naturally, when the functional disorder is associated with any degree of treatable organic disease, both factors must be adequately treated. When the

emotional disorder is felt to be primary, treatment consists of eliminating the functional symptoms and any underlying psychological abnormality.

Careful and complete history-taking and physical examination are important not only from the standpoint of what they may tell the doctor but also because of their reassuring effect on the patient, who will understandably be exasperated if his supply of information is not exhausted. The physician's manner is of great importance.

Patients with emotional disorders usually deny that they are nervous. The problem of the physician is to get them to realize the causal connection between stressful situations and physical symptoms. This may best be accomplished by setting the personal (biographical) history, which relates the stresses at various times, side by side with the history of past and present symptoms, and pointing out to the patient that the symptoms arose when he was most upset: of this the patient must convince himself fully. Prof. Farquharson[1] warns that the physician must be completely convinced of his diagnosis and confident in manner, for the worst thing he can do is to leave some doubt that the symptoms may have a organic cause.

The diagnosis and the mechanism of production of symptoms should then be explained to the patient in whatever detail is necessary to give him a satisfactory understanding of his illness; these explanations should be repeated as often as necessary, without the doctor's showing signs of annoyance. It is best to deal in principles, giving examples and analogies that are easily understood, such as the way in which emotion may produce such physical symptoms as blushing and nervous indigestion. Unnecessary detail should be avoided, although some patients require detailed explanation of what in their response causes symptoms (this is progressively more difficult the longer the emotional cause has been present). The intimation that emotional symptoms are due to disturbance of autonomic regulation rather than "all in his head" will usually give the patient solace, confidence, and self-respect. He must not only learn (in repeated sessions) to understand his symptoms, but he must also take an active part in training himself to become and remain well.

Later interviews should be undertaken as needed. While it is wise and economically necessary to have a flexible system, the individual interval between visits being dependent upon the need and the available time, most emotional disorders will respond to sessions lasting one-half to one hour, weekly or every other week. The tendency to relapse is remarkable, and it is therefore wise to arrange to see the patient again before the period of remission is expected to end. In subsequent visits, the diagnosis and explanation should be repeated as necessary, but not the physical examination (otherwise the patient will feel that the diagnosis is in doubt). When it is decided that the interviews may be terminated, it is wise to plan ahead, preparing the patient for this event, since he may have become dependent upon the doctor; feelings of rejection should be watched for, and dealt with as they arise.

It is important to remember that neurotic people are not immune to physical disease—the fact that the patient has been shown to suffer from functional disorders in the past should not obviate regular, complete, careful examination. We know of one case in which a stage II carcinoma of the cervix was discovered by a careful physician although none of the patient's thirty-nine typewritten functional complaints

was related to the disease (she was in fact averse to pelvic examination at first).

Treatment of an underlying neurosis may usually be managed by minor psychotherapy, by altering the stressful situation at the root of the difficulty if possible, or by altering the patient's reaction to the stress. Those patients in whom the emotional problems cannot be resolved by these measures must be referred to a competent psychiatrist. Other indications for referral are prolonged or dangerous depression, obsessive-compulsion (although the psychiatrist and the general practitioner may plan together), and those patients who are disabled or out of touch with reality. In this regard, the practitioner's approach to referral is important: referral requires time, tact, and the patient's confidence in both his own doctor and the psychiatrist. Except in cases of obvious emergency referral is better delayed than forced, for the subsequent management of the bullied psychiatric patient is considerably more difficult. The physician should, in these cases, complete the history, physical examination, and the necessary laboratory studies, but he should not give in to the patient's demands for unnecessary tests. When he suggests referral, he should be prepared to recognize and deal with the patient's feelings of rejection.

In many cases social management is important: the family doctor must work out a social programme and recreation for many of his patients, especially those suffering from emotional disorders. He may also have to educate the family to deal with the patient, or he may be called upon to alter stressful situations and to make arrangements with employers and ministers.

The results of treatment are excellent in patients of the first group, whose emotional stress causes only slight derangement of autonomic function: thorough examination and subsequent reassurance and explanation dispel anxiety, adequate rest contributes to quick recovery, and training prevents recurrence. Recovery may also be quick in patients of the second and third groups who are intelligent, co-operative, and desirous of being helped. A favorable environment is important; resentment and anger make treatment difficult. Similar results may be expected in dull, trustful people. Results are poorer but variable in weak, unstable, or psychopathic people, who are easily discouraged and believe whatever they are told by laymen in the interval between visits. Anxiety in a true depressive state is refractory, since it is a manifestation rather than a cause of the depression; similarly, fixed obsessions seldom respond to simple minor psychotherapy.

Conclusion

The general practitioner who is patient and understanding, who has a deep love of people and sympathy for their problems, can do much in the handling of very complex emotional situations. It is essential to the mental health of his patients that he have an appreciation of the importance, scope, and practice of effective psychological medicine.

In view of the high incidence of functional disorders, the student would be well advised to equip himself to recognize and to treat these problems adequately. This requires experience, of which he can obtain little before graduation, but he can maintain an awareness of the emotional problems to be discovered in all patients, he can learn to consider diagnosis as an evaluation of the contribution of both organic and functional factors in every patient, and he can try to develop that manner which is the art of medicine.

Increasing co-operation between psychiatry and the rest of medicine is already leading to several very desirable results: practitioners are more ready to call upon the psychiatrist when his special skills and training are needed, they are studying the psychosomatic approach and applying it to treatment in a more scientific way than previous generations, and they consider total diagnosis of all that afflicts their patients to be an ideal worth striving for. The logical outcome of these tendencies is that medical practice draws ever closer to Trudeau's enunciation of the simple, modest aim of the good practitioner . . . "To cure sometimes, to relieve often, to comfort always".

Summary

The general importance and scope of effective psychological medicine in general practice are considered. The causes, clinical features, and general plan of treatment of some common emotional disorders are outlined.

Acknowledgement

The author wishes to thank Dr. K. J. R. Wightman and Dr. Allan Walters of the Toronto General Hospital, both for their suggestions and for their permission to use material from their lectures in the preparation of this article. While it has not been thought practical to make specific acknowledgement of every point in the text of this discussion, it is expected that readers of Prof. R. F. Farquharson's excellent article[4] will readily appreciate the author's indebtedness to him.

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THE DOCTOR AND THE FAMILY OF THE RETARDED CHILD

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YOU are just about to see the parents of a three-year-old child who was brought to your office last week because of a delay in speech. On the previous visit you carried out a physical examination and found no organic cause for the absence of speech. You then referred the child for a psychometric and have just now received the report of the psychologist. This report indicates that the child has a mental age around twelve months and an intelligence quotient in the thirties. The parents with whom you are going to discuss your findings have, to the best of your knowledge, no idea that their child is mentally retarded.

You have found that this is unfortunately not a rare situation in your practice, because the incidence of severe mental retardation in your community is approximately one in every two hundred of the population. From your past experience you know that this diagnosis will be a severe blow to the parents, one which will shatter all their plans for the future of their child. They

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must face the fact that their child has a condition that is fundamentally incurable and which at the present time cannot be altered by any known procedure. It will persist throughout the life of their child and, since the advent of modern antibiotic therapy, will not appreciably alter the life span of the child. It will severely limit the education of the child, will prevent him from being self-supporting, from marrying or having a family of his own. It also forces the family to supervise his daily living, and in some manner provide financial support for him throughout his life. Parents are called upon to face few more traumatic experiences; even such diagnoses as incurable malignancy, leukemia, etc. are less traumatic in many ways, because in these diseases the problem remains one of months and not of a lifetime.

THE EFFECT OF THE DIAGNOSIS UPON THE PARENTS

You wonder how the parents will react to the diagnosis, knowing from your past experience that parents react to this in various ways. Some may be overwhelmed by the diagnosis and become depressed for days or even months. The degree of the depression varies, but on occasion may be severe enough to withdraw the family from the community for a period of time.

Some parents become anxious, some with overt signs of tension and anxiety, others with less obvious signs of tension. In these latter the tension may be revealed through various somatic disturbances: headaches, diarrhoea, nausea etc.

In some parents the diagnosis may arouse feelings of guilt, the parents in some way considering themselves responsible for the retardation in the child. Sueh may occur where there is a family history of retardation, or where the parents feel that some sexual activity or misdemeanour on their part has caused the retardation of the child.

Some parents deny the diagnosis and refuse to accept the fact of the retardation, even though it may be extremely obvious. In most cases this denial is of short duration, but it may persist for years in a paranoidal manner. On occasion one sees the parents of older children or even adolescents, who are incapable of accepting the fact that their child is grossly handicapped and retarded. Many of these parents go from physician to physician, from city to city seeking a cure, a different diagnosis, or a hopeful prognosis for their child.

In some parents the diagnosis arouses frank hostility, often towards the medical profession, which is blamed by the parents for the retardation of the child. They consider that it has been produced through faulty obstetrics, incorrect diagnosis, or inadequate medical care. At times this hostility to the medical profession is projected upon the unfortunate physician who first faces the family with the diagnosis. In other parents the hostility seems to be directed toward fate or towards God whom they feel have been grossly unfair to them.

You also know that most parents. in spite of their anxiety, tension, and feelings of depression, are able to accept the diagnosis, to face the problem with courage, and to make adequate and reasonable plans for the future of the child. However, you are well aware of the fact that, however calmly these parents appear to accept the situation, they are and must inevitably be deeply disturbed by the impact of the diagnosis.

You also wonder what will be the attitude of these parents towards their retarded child, knowing that parents have varying feelings towards these children. Some will overprotect the child, dedicate their whole life and plan their future around the needs of the

child. Others will have the opposite feelings, either open or hidden rejection, more frequently the latter. Such parents will be ashamed of their child, isolated from the community as far as possible, or request immediate placement in an institution. Again, from your experience you know that most accept the child in a realistic manner, and their feelings of overprotection or rejection are not sufficient to prevent them from working through an adequate programme for both the child and the family.

WHEN TO TELL THE PARENTS THE DIAGNOSIS

You realize this interview will be extremely traumatic for the parents and must be handled with the utmost tact and sympathy. You must have, if at all possible, adequate time to diseuss the problem in detail; in this regard you often find yourself in a better position than the specialist because of your previous knowledge of the family. You also know that parents will frequently become so disturbed at this time that they are incapable of remembering much of the interview, apart from the fact that their child is retarded and the future hopeless. For this reason it is often necessary to have a further visit at which time the parents will be less disturbed and can go into their problems in a more satisfactory manner.

You must also decide at this time whether to give a definite diagnosis and prognosis or to defer this until a further reappraisal. In the case of younger children and infants it has often proved wise to avoid a definite diagnosis at the initial examination and to bring the child back in from six to twelve months' time for a further appraisal and a more finite diagnosis. This delay in the final diagnosis serves several purposes. In the first place it provides a greater measure of accuracy and reduces the number of children in whom an erroneous diagnosis is made, and in the second place most parents are better able to accept the diagnosis if the child has been seen over a period of time. Many parents, particularly those of infants, feel that the physician or psychologist has not the ability to predict the future of the ehild on such a limited examination.

Where the final diagnosis is deferred you usually tell the parents at the end of the initial test the level of function of the child at the time of the test. In this three-year-old child you will tell the family that he is functioning at the present time as a twelve-month-old infant. This is usually something that the parents can accept, because they in their own minds have realized that the development of their child is delayed, and if they have had other children they have often been able to make a surprisingly good estimate on their own part of his level of function.

You will then tell these parents that it will be advisable to have a further test of the child in twelve months' time. The purpose of this is to see whether the present rate of development remains the same or whether the rate changes in the future. If in twelve months time the child is still functioning at the same rate then a final diagnosis can be given and an estimate made of his ultimate future. However, if there is a definite difference in the rate of development in these two tests it will be necessary to have further tests until the rate becomes stabilized and constant. The purpose of this is to provide a wider period of observation and to give a more accurate appraisal of the ultimate capacities of their child. However, as you feel that the child is likely to be retarded, you will prepare the parents at this time for the possibility of eventual retardation, saying that you suspect that this child is not going to

function as a child of normal ability but that no definite opinion can be given at this time and that you may be wrong.

You also realize that there are times when it is advisable to give a definite diagnosis at the time of the examination. While such a definite diagnosis will undoubtedly be disturbing to the parents it is often better for them to face the facts at this time than to continue in suspense and uncertainty. A definite opinion may be given in the case of infants where there is gross maldevelopment such as a microcephaly, etc., and in young children whose parents appear emotionally stable and indicate that they desire to know the ultimate future of the child in order to make realistic plans for him. It may also be given in the case of older children where the diagnosis is quite definite and in those children who have previously been diagnosed as retarded and where you are asked to give a second opinion.

You must also in the case of this three-year-old child decide whether to offer the parents a consultation or a second opinion. When one remembers that the diagnosis is a final one without the offer of any remedial measures it must be remembered that most parents will hesitate to accept the final decision of any one individual, no matter whether he be family physician, specialist, etc. A physician seldom loses stature in the eyes of the parents by suggesting such a consultation. It means that he is aware of their anxieties and is also willing to have his observations, conclusions and diagnosis scrutinized by any other physician. In cases where the diagnosis is made for the first time it is often of value to ask the parents if they would like a second opinion. If they so desire it is best to ask them to wait for a period of time for this opinion, but if they are incapable of doing so then it may be necessary to arrange an immediate consultation. Not all parents will want this extra opinion, and a large number will accept your diagnosis, particularly if you offer to them the possibilities of a consultation.

HOW TO TELL THE PARENTS THE DIAGNOSIS

When the time has arrived to frankly discuss with the parents the diagnosis and to outline to them the future of their child there are some practical points which you know will be of value.

You have found that in discussing the origin of the retardation it is often of value to point out to the parents that this condition is far from rare, and that anywhere from one-quarter of one per cent to one-half of one per cent of the community are born with similar degrees of mental retardation.

Where the retardation is of developmental origin it is often of value to compare this to such well known congenital defects as clubfeet, hare-lips, etc., stating that as in these other conditions the brain has not developed in a normal pattern and shows on examination deformities which affect its function and produce the retardation.

Where the parents ask whether the retardation is of hereditary origin it has proved of value to state, whether true or not, that in most cases of developmental retardation it appears as though the constitutional factors must be present in both the father and the mother. It is also of value to point out that in any family if a careful history is taken over several generations, the probability remains high that some retarded individuals will be found. These explanations reduce the feelings of guilt that occur in parents and the tendency of one parent to blame the other for the retardation.

Where the parents ask about the future of the child, experience has

shown you that it is often of value to discuss this prognosis in such practical terms as whether their child will walk, will talk, will be able to attend school, and if so the grade level which can be anticipated. They also wish to know whether the child can be trained to support himself, whether he will require guidance in later life, and whether he will possibly marry and have children. It is also of value to discuss with the parent the ultimate mental age of the child at maturity, and that more than anything else seems to give them a vivid idea of the ultimate capacities of their child.

You have been frequently asked about the advisability of having further children. Where there is no gross evidence of retardation among other members of the family, it is usually considered safe to have further children. The parents can be told that although the risk of having another retarded child cannot be estimated with precision. it does appear that the chances of their having such a child is not more than one in fifty to one in a hundred.

INSTITUTIONAL CARE OR NOT?

One of the most difficult decisions to be made by both the parents and yourself is whether this child should remain at home or be eventually placed in an institution for retarded children. Experience has taught you that this is a decision which in most cases is an individual one and must take into consideration the whole family—the child, the parents, and the brothers and sisters. Many factors must be taken into consideration in this decision and it appears that the most important are:

(a) the degree of retardation – the more retarded, the greater the need to institutionalize the child.

(b) the temperament of the child—the more disturbed, hyper-active, or disobedient the child, the greater the need for institutional care.

(c) the size of the family—in general, it is often possible to care for only children at home whereas such children in larger families may require institutional care.

(d) the health of the parents—physical illness on the part of the family, particularly the mother, must be considered as one of the indications for institutional care.

(e) the emotional health of the family—emotional disturbances in the family, again particularly in the mother, must be considered as factors suggesting institutional care.

(f) the effect on siblings—if the siblings are disturbed by the presence of the retarded child in the home it is often best to place him apart from the family. This is particularly true when the other children are in adolescence or at the age where they may be contemplating marriage.

This effect upon the other children in the family is most important and may be the major factor in the final decision. Parents will often keep these children in the home in spite of the pressures placed upon themselves by the child, but will allow the child to enter an institution if they feel that the retention of the child in the home will have deleterious effect on their other children.

It must be remembered that many parents have strong feelings against placing their children in an institution. In the first place they feel that it is their duty to care for the child and that they are shirking their responsibility when placing him away from their home. Some have feelings of guilt about the child, and wish to maintain him at home as an unconscious form of punishment to themselves. Some are anxious about the physical care of the child, believing that he cannot be given adequate care in a

large institution. Some doubt the ability of the institution to provide personal care for the child and to meet what they recognize as the obvious need for affection in the child. Finally, others feel that the child cannot reach his optimal level if he is living in a large "impersonal" institution, believing that the child can achieve this level only if he were to remain at home and were given an intensive, individually planned programme by the family.

All this has led to the recent move towards the home-care of the retarded child. At the present time a great attempt is being made to see if the child cannot adjust in his own home before considering institutional care. However, this in turn is creating a new problem, that of providing for the care of the child after the parents have become physically incapable of looking after him. In many cases this merely means institutionalization at a later date.

WHAT CAN BE DONE FOR THESE CHILDREN?

You realize that you will undoubtedly be asked by the parents what can be done for their child. While nothing can be done to alter the ultimate potentialities of the child, there are a number of measures which will assist him in achieving his optimal level in the community.

From time to time various procedures have been advocated for the improvement of the intelligence of such childdren. Some years ago glutamic acid was stated to produce a definite improvement in the intellectual ability and personality. Time unfortunately has shown that the changes, if any, are minimal and are of little significance in the ultimate future of the child. Similarly, surgical operations have been considered, amongst the most recent the production of an arterio-venous fistula between the internal carotid and jugular to improve the circulation in the brain. However, apart from the production of a coincidental hydrocephalus no significant improvement has been noticed in these children.

A great deal can be done for the child, even though grossly retarded, if his parents are capable of accepting his capacities. That means that the demands upon him will be compatible with his ability to fulfill those demands and that invidious comparison will not be made with the more normal children. It also means a programme planned for him which is based on his individual capacities and temperament, and a special programme of teaching, using technics that have been demonstrated to be of value for the retarded child.

You know that there are a number of pamphlets which are available and which give the parents some general instructions in the home care and training of these children.* These stress practical methods which will assist these children in learning the normal routines of the home and in developing normal habits. The pamphlets will also lead some parents into a wider range of reading which will in turn be beneficial to both them and their children.

In many centres there are Parents' Councils for Retarded Children, which are available to the parents of retarded children in that community. These organizations provide factual information for the parents, and also through group sessions give guidance and mutual support. These Councils have been of great value in obtaining facilities for retarded children and improving the lot of these children in the community.

In a number of cities psychiatric social workers are available to the parents of these children. These social workers may be attached to neurological and psychiatric clinics in the community, to the educational system of that community,

*See end of article.

or to the Parents' Councils themselves. These social workers assist the parents in working through their attitudes towards the child. They are primarily concerned in helping the parents clarify their feelings towards the child and helping them reduce their anxiety, depression, feelings of guilt, hostility, etc.

In some of the larger cities special nursery schools are now available for the grossly defective child. Their programmes are specially planned, and while there is a certain amount of instruction the major focus is on the socialization of the child. In addition to the benefits given to the child by the special programme, further help is given to the parents by reducing by a few hours the continual, almost never-ending burden that is placed upon them by the care of their child.

Since most Departments of Education do not accept the responsibility for the education of children with ratings below 50, and since most auxiliary classes will not accept these children, new classes are being planned to accept children who are grossly retarded. These classes are in a fact a continuation of the nursery school for retarded children, and like that nursery school the programme is partly educational and partly recreational and social. Such classes have shown that with skilled teaching the grossly retarded individual is capable of learning more than had been thought. Reading, writing, and simple calculations are within the capacities of a number of these children as well as a great improvement in their ability to adjust to their social environment. The value of such classes has now been proved, and certain provinces (e.g. Ontario) now make financial grants to the organizations that maintain these special classes.

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THE VOLUNTARY MENTAL HEALTH MOVEMENT IN CANADA

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IT has long been characteristic of our Canadian way of life that when a crisis or a social problem arises people try to do something about it. They seldom are content to wait for the machinery of official action to get rolling. They organize committees; they start to raise funds and they try their best to push the authorities into action with a minimum of delay. Perhaps this is partly due to a conviction, inherited from the frontiersman and the pioneer, that when a job

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needs doing, citizens should organize to get it done.

Something of this spirit has very obviously come into the health field. People—citizens of all kinds—are becoming increasingly concerned about health and are organizing to do something about it. For the most part this interest and activity is welcomed by the public health and medical authorities. The great advances which have been made in the fight against tuberculosis, cancer, and more recently arthritis and other crippling disorders have been possible largely because of this type of organized public support.

One of the oldest but least publicised of these voluntary health movements in Canada is that devoted to mental health. Organized in 1918 as the Canadian National Committee for Mental Hygiene, it has now become the Canadian Mental Health Association with active divisions in eight provinces.

To grasp the significance of a voluntary public movement such as this, we must understand something of the attitude with which mental illness has been regarded in the last 50 years. At the turn of the century in Canada there were only a few institutions providing care for mental patients. These "lunatic asylums", though designed, built and dedicated with great sympathy and humanitarian concern for the mentally sick, inevitably became the objects of political patronage. Public interest which helped initially to provide buildings for the custody of these "unfortunates" soon disappeared. Since mental illness was regarded as a hopeless affliction and since no one was aware of the true dimensions of the problem (there were no annual statistical reports until early in the 1930's) there was no particular public pressure to develop or improve treatment facilities. Shocking abuse and callous mistreatment of patients became widespread in these institutions throughout the country. During World War I the problem of "shell shock" aroused medical and public interest in psychiatry. Dr. C. K. Clarke, superintendent of the Toronto General Hospital at that time and Professor of Psychiatry at the University of Toronto, organized a psychiatric out-patients clinic in the hospital in co-operation with Dr. Clarence M. Hincks. It wasn't long before Dr. Hincks with Dr. Clarke's support had organized the National Committee. And the first assignment undertaken by this voluntary health organization was the survey of some of the provincial institutions which were caring for mentally ill veterans. This was in 1918. There followed thirty years of work consisting of consultations with provincial health authorities and surveys of mental institutions in all provinces in Canada. No public fund-raising campaign was held and no publicity was given to this work. The program was supported financially by the generosity of a group of public spirited citizens and some of the great philanthropic foundations.

During this period, the organization either directly or indirectly influenced the whole field of mental health in Canada. Institutions became mental hospitals, active psychiatric treatment services were established, Departments of Psychiatry and Psychology were organized in many universities, research was stimulated and financed, community mental health clinics and child guidance clinics began to function, and schools began to recognize and try to help those children who because of intellectual retardation or emotional disturbance were not succeeding in the classroom.

Since the Second World War the public interest has increased rapidly. Hardly an issue of the more important national magazines comes out without reference in some way to mental health and ill-

ness. And perhaps unfortunately, some of the cheaper pulp magazines are entirely devoted to subjects like "Personality" and "Human Relations". Books on mental and emotional health—often of the self-help variety—are among the best sellers. Radio and TV programs regularly carry dramatic programs and talks on this subject. By no means is this publicity always good or useful. But it certainly reflects public interest and possibly also public concern.

It is an interesting fact however that with all this activity there is still a great deal of prejudice and misunderstanding about mental illness. Such disorders are frequently regarded as shameful, as being directly inherited, as being a kind of visitation or punishment for sins in the past and as being hopeless and untreatable. None of these ideas of course is true. But the level of superstitions, fear and misconception is so high that mental hospitals are still regarded by many people as curious and frightening places to which people sometimes have to be sent and from which they seldom return.

This situation has been a challenging one for the voluntary organization and new ways and means have been sought to cope with it. In 1951 the National Committee became the Canadian Mental Health Association. Gradually, divisions of the association were established in many of the provinces. This had the effect of providing the layman with both the stimulus and the opportunity of participating in the program.

Meanwhile the Federal Government had established substantial "mental health grants" which were designed to assist the Provincial Health Departments in the training of professional personnel, and the extension of treatment services. These two developments—the growth of the voluntary association and the establishment of mental health grants—have had a very important effect on the mental health program in our country.

Today there are about 66,000 patients in our mental hospitals. They occupy at any one time about half the hospital beds in the country. The number of physicians and ancillary professional staff employed in their care and treatment has steadily increased. Psychiatry is now well established as one of the important medical specialties for which postgraduate training is offered in several Canadian universities and certification is granted on examination by the Royal College of Physicians and Surgeons of Canada. A challenging career as a specialist in this field either within the provincial hospital organization, or in private practice is now assured for those who are interested and can qualify.

The Canadian Mental Health Association is reflecting this increase in momentum in a variety of ways. Only two examples of current program activity will be cited here as having special interest to the medical profession—the Volunteer program and the Training Program.

While volunteer visitors and workers have long been accepted in general hospitals, it is remarkable that organized visiting in mental hospitals by volunteer groups has only developed in this country in the last few years. This has now become a highly organized and valued part of many mental hospital programs. Problems relating to the selection and training of volunteers, their various assignments in the hospital and the evaluation of the service are being worked out. These visitors not only provide social contacts for the patients, many of whom have been forgotten long ago by relatives and friends alike, but they carry out projects with the patients which have significant therapeutic value. Such projects include assisting with the pat-

ients' library, providing instruction in basic English, holding sing-songs with the patients, helping with walking patients on shopping excursions and even serving as a liaison with the patient's family. This activity serves to bring more and more people from the community into touch with the mental hospital and of course brings the hospital closer to the community. It helps to compensate for the unfortunate fact that our mental hospitals are for the most part located quite a distance from the large urban centres.

A new aspect of the volunteer visitor's work is the rehabilitation program which has already been started in one or two provinces. This, of course, is a natural outgrowth of the contact with the patient in the hospital. The friendly interest and help of the volunteer continues after discharge. In one province there are plans for a CMHA rehabilitation centre or a kind of club for discharged patients where they can feel welcome and receive social and emotional support. A professionally qualified social worker will be on hand assisted by the volunteers.

The Training Program of the CMHA is quite different. This training does not attempt to provide basic professional qualifications which is the responsibility of the universities. But it does serve as a means by which short informal training experiences for different professional groups can be established. These "training experiences" vary from the short one- or two-day institute or "workshop" for nurses or social workers, to the rather elaborate academic year's training worked out in collaboration with the University of Toronto for teachers. These training projects are not designed for degree purposes but only to help these groups of professional people become more alert and perhaps a little more skilful in dealing with the mental health problems encountered in their regular work. In the case of the special course for teachers at Toronto the purpose behind the project is to bring to the University a carefully selected group of teachers from across the country who are exposed to a variety of courses from different professional fields including psychiatry, sociology, social work and psychology. On their return to their own schools it is hoped that they will be given an opportunity to serve at least for part of their time as a kind of consultant to other teachers with reference to mental health problems in the classroom. They are not regarded as clinical psychologists or social workers but simply as teachers with hopefully a deepened interest and insight into children's behaviour and mental health.

This program was started in 1949 and to date over 40 teachers representing every province in Canada have been trained. A careful evaluation study of the project financed by the Carnegie Corporation has been made and, as a result, the course is now formally included in the University's post-graduate department.

The value of the visiting program in helping with the recovery of patients and the value of the teacher training project in preventing mental illness through the early detection of cases and the establishment of a sound mental health environment in schools has already been substantially demonstrated.

The activities of the CMHA are quite extensive of course, and these two examples—one illustrating the role of the lay citizen and one the opportunities for the professional person—are merely indicative. It is the firm resolve of the people who constitute this organization to work patiently but persistently towards the establishment of better diagnostic and treatment facilities for the mentally

ill and the development of a sound protective and preventive program in mental health for all!

Further information about the program can be obtained from the national office of the Canadian Mental Health Association, 111 St. George Street, Toronto, Ontario. Information on training opportunities for graduate medical students in Canada can be obtained from the Royal College of Physicians and Surgeons, Ottawa, or the Dept. of Psychiatry, University of Toronto.

CASE REPORT HUNTINGTON'S CHOREA

T. D. R. BRIANT, 5T5

RUSSELL BRAIN defines the disease as a hereditary disorder characterized pathologically by the degeneration of the ganglion cells of the forebrain and corpus striatum and clinically by choreaiform movements and progressive dementia which usually begins during early middle life.

It is a relatively rare heredo-familial affliction whose incidence may run as high as ten to twelve thousand cases in North America. The disease is very rare in Britain, and most of these cases were limited to certain families of the fishing village of Avoch in Ross-shire. A high percentage of cases in America studied by Muncey and Davenport were traced back to six individuals. It is believed that the disease was introduced to America by three immigrant brothers who settled in the areas of Boston and New Salem, Massachusetts. Both Avoch and the American ports were colonized in the early seventeenth century by Puritan stock from Boston in Lincolnshire, England.

Although the disease was described as early as 1635, it was George Huntington, a Long Island practitioner whose father and grandfather had practised in the area, who described the disease and realized the importance of the heredofamilial element. Since then it has been found to be transmitted directly from one generation to the next. It seldom skips a generation, unless the parents die before the age of onset, selecting about half the children of any one generation. The usual age of onset is between 30 and 45 years but wide variations have been recorded, ranging from early childhood to the seventh decade.

Pathology The basic lesion is that of a degeneration of the ganglion cells and extensive proliferation of neuroglia. This may be seen in cortex, basal ganglion, cerebellum and occasionally in the spinal cord; the sites of predilection are, however, the corpus striatum and the frontal areas of the cortex with sparing of the temporal and occipital poles. The brain diminishes in size and weight and the convulutions become small and atrophic.

Classical Features (a) insidious onset of involuntary movements being most conspicuous in the face and hands leading to dysarthria and ataxia of the limbs. (b) mental changes which develop subsequent to the chorea, consisting of a progressive dementia in which the patient becomes inert, apathetic and irritable.

As the clinical picture does vary in different family strains, and indeed within a single affected family, Davenport and Muncey described four clinical types which might be of use in the recognition of the disease.

1. Tremors followed by dementia

2. Tremors with no dementia
3. Cases in which the chorea is not progressive
4. Cases seen early in life, in children of two affected parents.

A further type is described by Curran who reported four cases in which chorea was absent or of very late onset, but with marked mental changes.

The first symptoms in cases featuring dementia may be increased irritability, malaise and insomnia. Individuals in which dementia is absent or slow in appearing the first premonition of the disease is an insidiously developing chorea.

Where dementia is present the affective side of the patient's personality is altered to the greatest extent. Commonly there is a manic depressive picture which may go on to complete loss of emctional expression. Ravenous appetite and lack of insight into the disease are common. Suicide is relatively rare probably due to this lack of insight.

The patient's thinking processes are not involved to the same degree as is the affective side of his personality. He may be able to answer specific questions with reasonable accuracy, although as a rule there is some impairment of intellect in the spheres of memory, concentration, calculation and especially judgment.

The chorea usually progresses until there is a marked dysarthria and ataxia. This is often so severe that the patient may become bed ridden. The duration of the disease is usually ten to fifteen years; the patient finally succumbs to secondary infection.

This article was prompted by the fact that greater numbers of these families are being seen in this district. Murray Hall recently described a family in which the diagnosis had been made in 1947. Recently two families with Huntington's chorea were seen within a week of each other at the Outpatient Department of the Toronto General Hospital. The following is a brief account of one of these families.

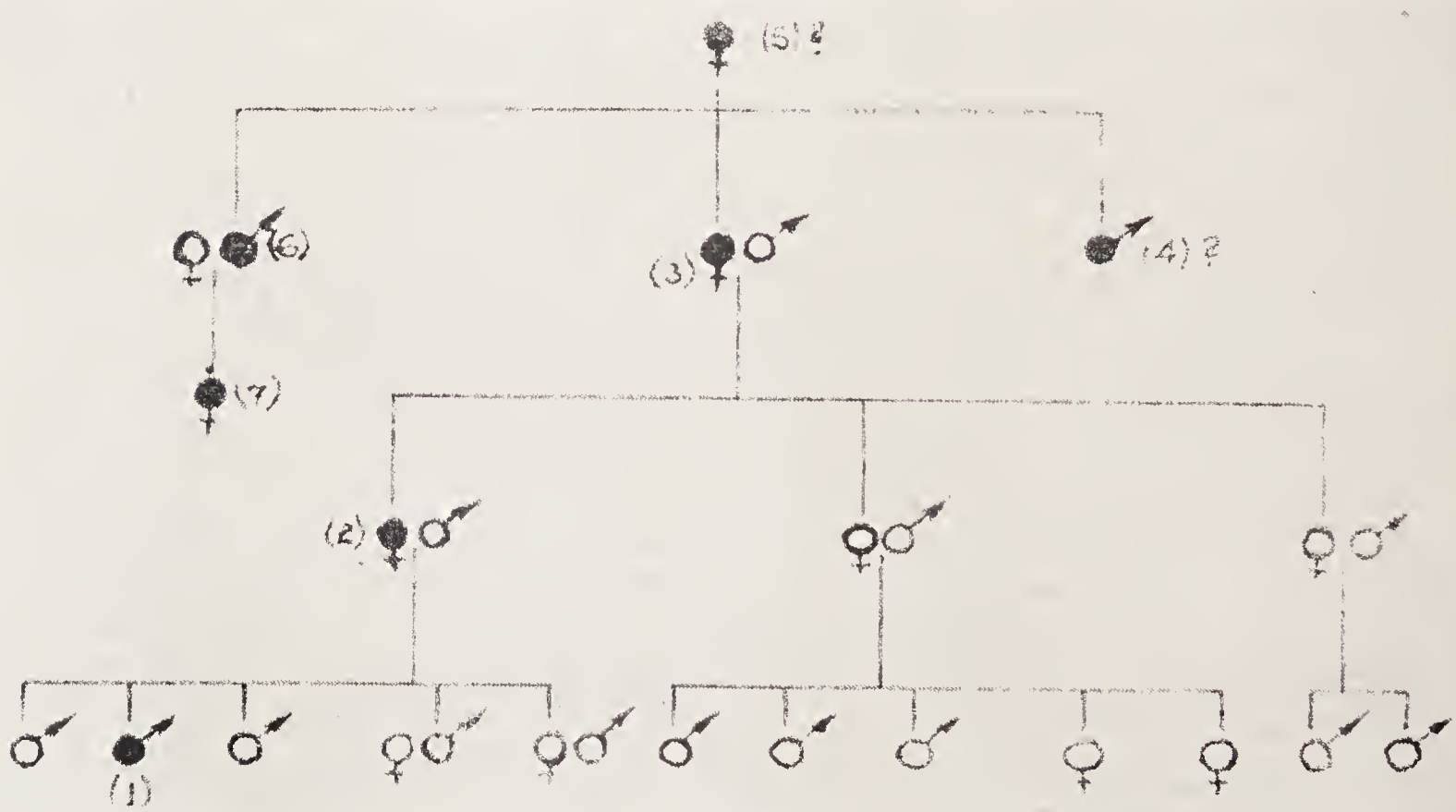

FIG. 1.—Genealogical Chart for case No. 1 indicating members affected and possibly affected.

A FAMILY HISTORY—(see fig. 1)

Case No. 1 The patient, a 32 year old single male machinist, was brought for examination by his sister who had noted a change in his personality and gait for one year. The patient himself had no complaints but followed his sister's lead meekly. The family noted that the patient who had previously been neat and tidy in appearance had become sloppy and cared litle for his appearance. Two years previously he had had many and varied interests; he gradually became less active and spent great portions of his spare time staring into space. A person who formerly drank moderately, he now spent great portions of his time in the beer halls and managed to squander a good bank account. He had been arrested by the police on several occasions for insobriety but curiously enough at times when he had not been drinking. The patient had always worked and enjoyed working at his trade as a machinist. Three months before being seen he was laid off following a number of incidents: he ran a drill press through his fingers on several occasions and walked off his job for hours at a time without permission. He was generally slow in performing his job. He set fire to a chair while smoking.

In the last year, his gait had become stiff, his face constantly twitching into a fatuous grin. He had become almost totally dependent on his family who gave him his meals, washed, bathed and shaved him. He developed a typically ravenous appetite. He voided only once daily and this is perhaps an unusual feature of the condition.

On examination, the patient was a well-nourished, well-developed male who appeared strangely youthful. His face was constantly screwed into a meaningless grin and his eyelids were half closed giving him a rather sleepy appearance. He was aware of his surroundings but they had no emotional impact on him. His intellectual functions were affected to some degree; he did not know the day or the month but knew the year; he had difficulty in counting backwards from ten. Emotionally he was flat: he could not be annoyed and occasionally gave a forced, mirthless laugh. His behavious was completely passive. The gait was stiff with no swing of the arms. Speech was often slurred and at times explosive in character. There was no muscle wasting; tone was variable on different days. The reflexes were all very brisk but equal and there normal plantar responses. There was minimal but definite left-sided dysdiadochokinesia. There was no sensory loss.

Case No. 2 At age 42, the mother of the above patient began to show chorea. Soon afterwards, she became irritable and then gradually demented. She was seen at a Psychiatric Hospital where her mental state was assessed. This showed that her memory, ability to calculate, and other intellectual faculties were reasonably well preserved. The affective side of her personality was altered: she was irritable, abusive and did not behave appropriately. The family noted that she too had a ravenous appetite and would go two days at a time before passing a large volume of urine. The patient gradually became ataxic and in the latter years of her life was confined to bed. She died 16 years following the onset of the disease.

Case No. 3 Little is known of the grandmother other than that she was said to have been affected similarly to the mother. She was bedridden in her later days and died approximately fifteen years after the onset of her condition.

Case No. 4 The great uncle of the patient was thought to have been affected. He had always been an ardent hunter and apparently shot himself while hunting. It was not known whether this was suicide or an accident.

Case No. 5 The great-great-grandmother was known to have some affliction in which she went to bed for the last ten years of her life and was nursed by the grandmother.

Cases No. 6 and No. 7 My informant believed that there were two other members of the family affected in these two generations.

Discussion

The first patient (case No. 1) demonstrates many of the classical features of the disease. The history of the mother and the rather suggestive history of other members of the family establishes the diagnosis. Certain types of presenile dementia occur in a family but they do not show the strong familial incidence of Huntington's Chorea.

At the present time there is no specific treatment for this condition: adequate nursing care and protection are the only means of management.

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ABSTRACTS AND REVIEWS

Clinical Manifestations of Temporal Lobe Epilepsy and their Recogniton in Relation to Surgical Treatment: Falconer, M. A., British Medical Journal, Oct. 23, 1954.

Temporal lobe epilepsy accounts for about one quarter to one third of all epilepsy. It is particularly liable to be associated with personality disorders and is often intractable to medicinal therapy, demanding surgical intervention.

The anatomy and physiology of the temporal lobe are outlined and the clinical contents of temporal lobe seizures are discussed. The factors are: (a) The situation of the epileptic focus within the brain stem; and (b) the violence of the local epileptic discharge and the extent to which it spreads not only within the temporal lobe, but to other regions of the brain.

The sensory and affective phenomena of the auras of temporal lobe epilepsy are discussed: (1) crude hallucinatory phenomena concerned with sensations within the temporal lobe. These include: epigastric sensations ("the stomach turning over"), choking feeling, unpleasant sense of smell or taste, tinnitus, disturbance of balance and crude visual hallucinations. (2) emotional phenomena manifested by a sudden change of mood such as fear, anger or bewilderment. (3) hallucinatory states based on memory mechanisms described by Jackson as the "dreamy state", in which the patient may experience a sudden famili-

arity or strangeness with his surroundings. More elaborate 'deja vu' phenomenon is one in which the patient feels he is witnessing or participating in a scene in which he has taken part before. (4) auras arising from adjacent regions of the brain, sueh as twitching movements or unilateral tingling or numbness, suggest involvement of sensorimotor areas above the Sylvian fissure.

Three types of seizures are reeognized: (1) minor seizures without loss of eonseiousness, (2) amnesic attacks without eonvulsions, and (3) major convulsive seizures.

Possibly half of all patients with temporal lobe epilepsy exhibit severe personality disorders during the inter-seizure period. Some patients experience a feeling of aggressiveness or hostility, while others exhibit anxiety states, depressive states and even schizoid states.

Broad principles of treatment of temporal lobe epilepsy are outlined:

(1) The recent appearance of temporal lobe seizures asociated with neurological signs or raised intercranial pressure suggests cerebral tumour, which deserves appropriate studies.

(2) In cases in which fits are of long standing and there is nothing obvious in the clinical picture to suggest tumour, control of the seizures should be attempted by drugs: Phenobarbitone, Dilantin and primidione.

(3) In cases in which the fits cannot be properly controlled, are disabling, or in the presence of severe personality disorder, full neurological, radiological and EEG investigations should be made. In a patient with intractable epilepsy, the EEG demonstration of an epileptic focus which is confined to one temporal lobe, or if bilateral predominantly, on one side in the author's opinion, is an indication for operation.

I. G. L. Van Praagh

The Psychological Implications of Overeating: Conrad, S. W., Phychiatrie Quarterly, April 1954.

When we realize at least 1/5th of the U.S. population over the age of 30 are 10% or more overweight, it is apparent that obesity is one of the more serious health problems of our time and, in this article, Dr. Conrad discusses the phychological implications of overeating.

Except for rare isolated cases such as encephalitis with damage to the hypothalamus or at the eultural level in certain societies, overeating is a symptom which implies an emotional problem.

The implications of overeating are divided into three causative categories: (1) anxiety, (2) gratification, and (3) hostility.

Anxiety Here the overeater is orally fixated and as his anxiety increases he regresses in an effort to find relief from his tension. This is an escape associated with feelings of insecurity and such events as a new job, the death of a loved one or a new marriage are examples of stressful situations which could produce the neeessary anxiety. To the person from a home where food was offered as a substitute for love, food may represent the only gratifying security this person understands.

Gratification This is the discontented person inclined to self pity who argues "if life is not good to me then I will be good to myself." In the already obese person diseouraged by ineffect of diet this can occur.

Hostility Conrad says this is overlooked too often in the supposed placid jelly fat person, but this is because it is so well repressed. He further divides Hositility into three forms:

(1) *The Defiant.* Deprived of love and rejected who will assert their independence and "get even" by overeating.

(2) *The Repressed Hostility.* Because the individual has a fear of retaliation associated with a desire to please. Here if the anger or resentment can be uncovered than overeating will usually cease.

(3) *Denial of Hostility.* By overeating to become ugly and prove that one is not a competitor in the game of life.

Diet and the use of drugs is notoriously unsuccessful in treatment, since these are only useful adjuncts. We can solve the patients ploblem only by employing psychotherapy and discovering the underlying causative mechanisms.

D. Gordon

Hereditary Vascular Tumours of the Nervous System: Maurice L. Siver, J.A.M.A., *156*, 11, 1053–8, (Nov. 13, 1954).

Also called Lindau's disease, manifested by cystic hemangiomata of cerebellum associated with angiomatous retinae (Von Hippel's Disease) and spinal cord (Wyburn-Mason) and less frequently cysts of lung and pancreas, cavernous hemangiomata of the liver, angiomata of the epididymis and often hypernephroma of kidney.

Lindau believes the disease is due primarily to the development of a vascular mesenchymal plate covering the 4th ventricle occuring during the third foetal month, at which time vascularization of retinae etc. is occuring. Neoplastic tendency is explained as a congenital rest proliferation.

The disease is transmitted as a Mendelian dominant, is not sex linked and is one of few inherited neoplasms. Its age of manifestation and organ involvement is quite variable.

Diagnosis is made upon the demonstration of two such vascular lesions in one patient (e.g.: unilateral blindness with fundascopy; cerebellar malfunction with increased intercranial pressure with ventriculography and vertebral angiography; hemiparesis and paraplegia, abdominal mass and hematuria) or the presence of a single vascular lesion in a patient with verified family history of the disease.

Treatment depends upon the location and growth potential of the tumours. In the retina palliation can be effected by electrocoagulation; in the cerebellum, only surgery is effective; in the spinal cord, extramedullary lesion respond to surgery, intramedullary lesions to irradiation, but results are equivocal.

D. W. Wilson

A Study in Juvenile Sex Offenders: Atcheson, J. D. and Williams, D. C. The American Journal of Psychiatry *3*, 366–370, (Nov. 1954).

Some conclusions are drawn from a statistical survey of all the sex offenders appearing before the Toronto Juvenile and Family Court between 1939 and 1948. In this period 3,112 juvenile delinquents were referred to this clinic, 283 being sex offenders, 116 boys and 167 girls. The findings were compared with a randomly selected samples of other offenders.

The following points are of interest: (1) Nature of the Charge: Specific charges involved male sexual deviations, whereas nonspecific charges usually implied promiscuity in female and normal sexual curiosity in the male. (2) Incidence: In the juvenile delinquent population there were more girls than boys charged with sexual misbehaviour. (3) Age: 62.9% of male and 87.5% of female delinquents were between 13 and 16 years inclusive. Compared with the control group sex offenders were older than non sex offenders. (4) Intelligence: There is no specific difference in IQ between sex offenders and

non sex offenders. (5) Socio-Economic Stress: There is no significant difference in the two groups. (6) Recidivism: A juvenile sex offender rarely reappears on a second charge at least up to age 16.

The following classification of juvenile sex offenders is considered: (1) Emotionally disordered sex offenders whose sex behaviour is a symptom of profound personality maladjustment: They are most frequently male and continue to create a problem in adult life. (2) Defective sex offenders. Males are slightly more prone than females. (3) i) Normal heterosexual female delinquents are promiscuous in an attempt to gain status, affection and security. ii) Normal heterosexual male delinquents. Their acts are usually based on a developing sex curiosity.

These problems demand inpatient diagnosis and treatment centres for children.

I. G. L. Van Praagh

Results of Lobotomy in Chronic Psychosis: Diamond, L. S., and Stauffacher, J. C.; Diseases of the Nervous System, *15*, 282–284, (September) 1954.

In 1949, Veterans Administration Hospital, American Lake, Washington, began selecting for prefrontal lobotomy, psychotic patients who were refractory to all of the usual treatments, and who were a burden to the programme of hospital treatment because of their inability to co-operate. Eighty-six per cent of those patients selected had been at the hospital for at least five years and appeared destined to indefinite confinement.

In a series of 77 such patients, each person was followed post-operatively for one to five years. Several patients showed improvement only after 6–12 months had elapsed, whereas several others demonstrated marked initial improvement but then a gradual return of symptoms.

The overall results in the group of 77 are tabulated. Eleven improved sufficiently to go home, or to remain at hospital with ground privileges. Fifteen now need only minimal supervision, either in the hospital or at home. Forty patients were partially relieved of symptoms, but still require a moderate degree of attention. Eleven showed no change whatever, but none became worse.

The commonest pre-operative behaviour problem had been assaultiveness, affecting 49 of the patients. Following operation, 31 were greatly relieved of this symptom, 17 were moderately relieved, and one showed no change. Good results were noted also for the characteristics of agitation, impulsiveness, and hostility. The operation proved to be less effective in cases of active resistiveness, passive restiveness, delusions, and hallucinations.

A. Goldberg

OF CURRENT INTEREST

During the second week of November, the University Christian Mission sponsored four noon-hour addresses in the new Medical Common Room. The Rev. Michael Creal, rector of the Anglican Church: "*Security*"; Dr. C. P. Martin, chairman of the Department of Anatomy, McGill University: "*Does the Gospel Need Reformation to Correspond with Scientific Knowledge*?"; Dr. G. G.

Harrop, assistant professor of Biblical Studies, McMaster University; "*Revelation and Authority*"; and Mrs. C. W. Sowby, graduate in Theology, Oxford: "*A Christian View of Marriage and Family Life*".

Noon hour films on subjects related to various medical courses are shown by Irv. Gollish, Jr. L. C. O., every Wednesday noon at the Banting Institute.

Those using the Interne Placement Service are reminded of the deadlines to be met.

The deadline for obtaining a C.A.M.S.I. Insurance Policy without a medical examination was December 31, 1954 for all years. For amounts over $10,000 and policies taken out after the deadline, a medical will be required. The rates are $3.00 per $1,000, and the plan is available to all C.A.M.S.I. members of the medical years, internes, and post-graduate students (who have not practised in excess of three months in any one year since graduation).

Faculty of Medicine

The Phi Delta Epsilon Fraternity Lecture was delivered by Dr. Alexander S. Wiener, Chief of the Transfusion Services in the Brooklyn Jewish Hospital, on November 15th, the subject of his lecture being: *Recent Advances in the Rh Field.*

The following awards have been made:

The R.C.A.M.C. Womens Auxiliary Bursary:
Miss P. E. Hanley (I Med. Yr.)

The C. Roy Sproat & Katherine Sproat Bursary: C. L. Berlie (IV Med. Yr.)

The Phi Delta Epsilon Bursary:
M. E. Miller (IV Med. Yr.)

The attention of the undergraduates in Medicine is drawn to the Loan Fund operated by the Medical Alumni Association. Students needing assistance of this character should apply to the secretary, Mrs. Velma Macfarlane, Room 12, Medical Building.

Daffydil 1954

The drums rolled, the audience arose, but it was a false start on God Save the Queen, and Daffydil 1954 was underway.

Epistaxis, making its second appearance since 1940, gave rise to but two reactions, only one of which was approval. This spritely little publication had a remarkably similar savour to its 1940 predecessor which went caput!

The theme of Daffydil '54 was the quest of two medical students, Bill Hunter and Dick Nero, III Meds, for a cure for baldness. Their search carried them to the intersection of Queen and Yonge, an operating room amphitheatre, and the high seas. At times the theme seems to have slipped right out of Hart House Theatre, but always it was recaptured, usually ingeniously, and just in time.

The interacts, again the responsibility of the premedical years, achieved their purposes to the full. There was never a dull moment as the premeds made enough shatter and clatter to keep the audience oblivious of the time-outs for set changes. More than a few maidens in the audience found themselves being carried by T.H.H.S. (Trans-Hart-House-Stretcher) to the stage, and there auctioned to the highest bidder who might have said anything from 10,00 rubles to six no trump.

First Meds' skit centered around the old, but for some reason always fresh, operating room scene.

Second Meds used the form of the tragic opera; eighteen persons met their demise, and lay strewn upon the stage. The Yonge St. subway, the St. Lawrence Seaway plan, and the partying indulged in by Toronto's ex-mayors of the past

few years were treated with jest. A well known (not the Royal York) Toronto hotel also met the full force of 5T7 satire.

Third Meds' skit was in a class by itself at least up until the Wednesday evening performance. The setting was a spaceship, and the theme the reactions of men without women when days turn into weeks and weeks turn into months, etc. Dodie Sperry's performance as the Lonesome Gal was a highlight of the skit.

The graduating year took this opportunity to blow up to gigantic proportions the habits and foibles of faculty members. The scene was set first upon a raft and then within the belly of a whale. Drs. Grant, Farquharson, Ham, and Hamilton were again the favourite targets of good natured but pointed exaggeration. Jim Ainslie, the image of his father, Dr. D. S. Ainslie, Professor of Physics, was magnificent in his portrayal of the kindly old master of the bar-bells. Drs. Best, Greey, and Price, and Dean MacFarlane completed this sea-going crew.

Credits for Daffydil '54 must go to one and all, from stage crew to performers to writers, but especially to Paul Steinhauer 5T7, Paul Clarke 5T8, Jim Rumball 5T9, and Ray Levin 5T9 who wrote most of the music and the hundreds of lines of lyrics. On behalf of the audience, we extend our heartiest thanks to directors Murray Herst 5T5 and David Sackin 5T5 for master-minding a show to remember.

A. C. STRICKLER

Licence to Practise in Ontario

The Medical Council of Canada has recently passed a regulation under which a candidate may not effect full registration with the Council until (a) he has passed the examinations; and (b) he has produced satisfactory evidence of having completed a year's interneship in a hospital which is approved by the province in which he proposes to register for practice.

This regulation will be in force in all the provinces of Canada, except Ontario, on January 1, 1955. The College of Physicians and Surgeons of Ontario have decided that they will not ask for the certificate of interneship until January 1st, 1958.

It is expected that a list of the successful Ontario candidates in the examinations of the Medical Council of Canada will be forwarded to the College of Physicians and Surgeons of Ontario, and they will be enabled, if they desire, to register with the College immediately. This arrangement, however, will cease on January 1st, 1958.

J. A. MacFarlane, *Dean.*

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Each Quadrinal Tablet contains ephedrine hydrochloride ⅜ gr., phenobarbital ⅜ gr., theophylline-calcium salicylate 2 gr. and potassium iodide 5 gr.

Quadrinal Tablets are marketed in bottles of 100, 500 and 1000.

Quadrinal, trade-mark registered; distributed by BILHUBER-KNOLL CORP., Orange, N J.

MERCK & CO. Limited, Selling Agents MONTREAL

OPTIMUM BARRIER without a diaphragm

ACTIVE INGREDIENTS BY WEIGHT
MONOLAURIN 5%, BORIC ACID 1%, GLYCERINE 1%, NONOXYNOL 5%
NET WEIGHT 3 OUNCES
DISTRIBUTED BY JULIUS SCHMID (CANADA) LTD. - TORONTO, ONT.
ACCEPTED
Ramses
VAGINAL JELLY

FASTEST IMMOBILIZATION

(measurable by officially accredited test)*

occlusive action lasting 10 hours

*Brown and Gamble technic

The word RAMSES is a registered trademark.

JULIUS SCHMID (Canada) Ltd.

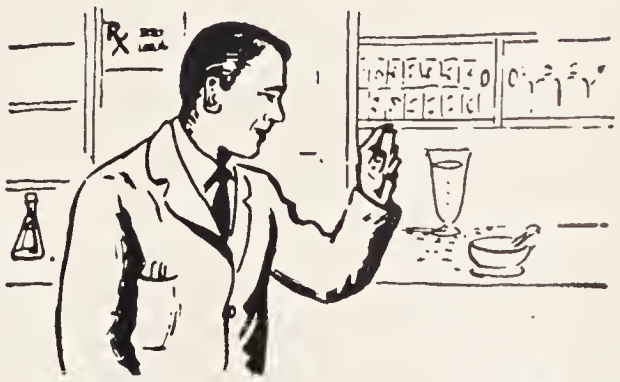
32 Bermondsey Road
Toronto, Ontario

Are you a stranger in your own home

due to the unending demands of your practice?

Time now spent running a dispensary could be better used for family activities.

Save this time by using the dispensing service offered by the pharmacist;



your full-time assistant giving you complete, dependable aid in all things pharmaceutical.

ONTARIO RETAIL PHARMACISTS ASSOCIATION

THE ONTARIO RETAIL PHARMACISTS' ASSOCIATION

"Noctinal"

Brand of Butabarbital NNR

In small doses, Noctinal reduces sensitivity to irritating external stimuli without seriously impairing efficiency.

The larger doses are more suitable for night-time sedation.

Tablet No. 439 "Frosst" ¼ gr. (16 mg.)
Tablet No. 352 "Frosst" ½ gr. (32 mg.)
Tablet No. 419 "Frosst" ¾ gr. (50 mg.)
Tablet No. 353 "Frosst" 1½ gr. (0.1 G.)

DOSAGE: ¼ to 1½ grains, three times daily.

Packaged in bottles of 100 tablets.

ELIXIR No. 601 "Frosst"
Each teaspoonful (3.5 cc.) contains:
"Noctinal" . . . . . ½ gr. (32 mg.)

DOSAGE: Adults: ½ to 3 drams (¼ to 1½ gr.) three times a day in a wine-glass of water. Children: according to age and physical condition.

Packaged in bottles of 16 fluid ounces.

Frosst

Charles E. Frosst & Co.
MONTREAL CANADA

Can you get away for some fishing, Doctor?

Or, as well as looking after a busy practice, are you doing your own dispensing with all its attendant details

keeping stock — compounding prescriptions — and bookkeeping? Prescribe through the pharmacist, your assistant trained to give you complete, dependable service in all things pharmaceutical

Precious time thus saved is better spent in other pursuits.

Good fishing, Doctor!

3

ONTARIO RETAIL PHARMACISTS' ASSOCIATION

THE ONTARIO RETAIL PHARMACISTS' ASSOCIATION

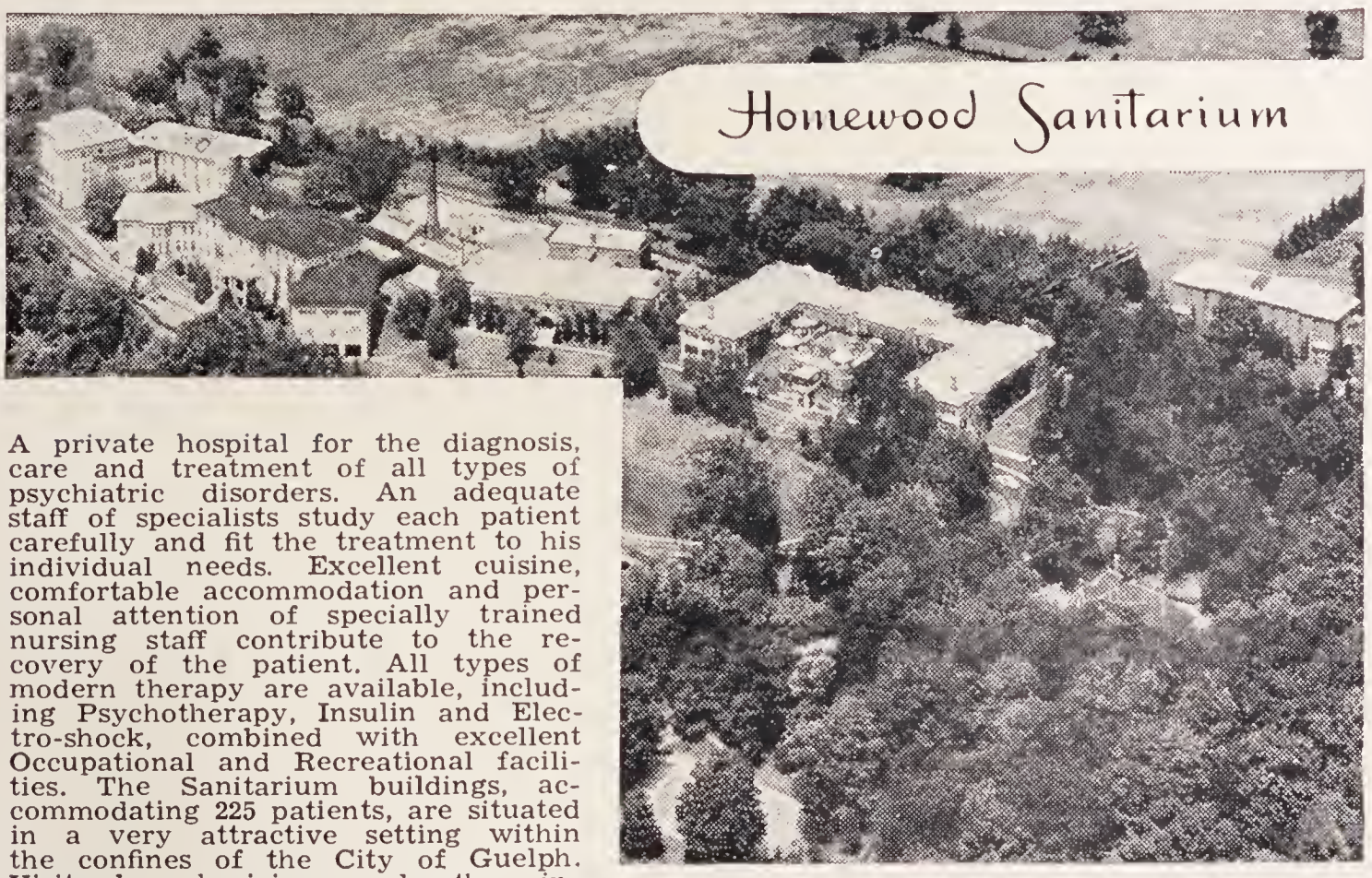
PROCEEDINGS OF THE FIRST WORLD CONFERENCE ON MEDICAL EDUCATION, LONDON, 1953
Held under the Auspices of the World Medical Association.
Edited by Hugh Clegg.
1954. 820 pp. $9.00

THE ANATOMY OF THE BRONCHIAL TREE
By R. C. Brock.
2nd edition 1954. 251 pp., 158 illus. (17 in colour) $6.75

BACTERIOLOGY FOR MEDICAL STUDENTS AND PRACTITIONERS
By A. D. Gardner.
4th edition 1953. 280 pp., 31 illustrations. $2.00

A THOUSAND FAMILIES IN NEWCASTLE UPON TYNE:
An Approach to the Study of Health and Illness in Children
By James Spence, W. S. Walton, F. J. W. Miller, *and* S. D. M. Court.
The Nuffield Foundation and The Nuffield Provincial Hospital Trust.
1954. 232 pp., 8 figs., 56 tables, 6 plates. $1.75

OXFORD UNIVERSITY PRESS



A private hospital for the diagnosis, care and treatment of all types of psychiatric disorders. An adequate staff of specialists study each patient carefully and fit the treatment to his individual needs. Excellent cuisine, comfortable accommodation and personal attention of specially trained nursing staff contribute to the recovery of the patient. All types of modern therapy are available, including Psychotherapy, Insulin and Electro-shock, combined with excellent Occupational and Recreational facilities. The Sanitarium buildings, accommodating 225 patients, are situated in a very attractive setting within the confines of the City of Guelph. Visits by physicians and other interested parties are always welcomed. For further information write or telephone (Guelph 53).

A. L. MacKINNON, M.B, Medical Supt.

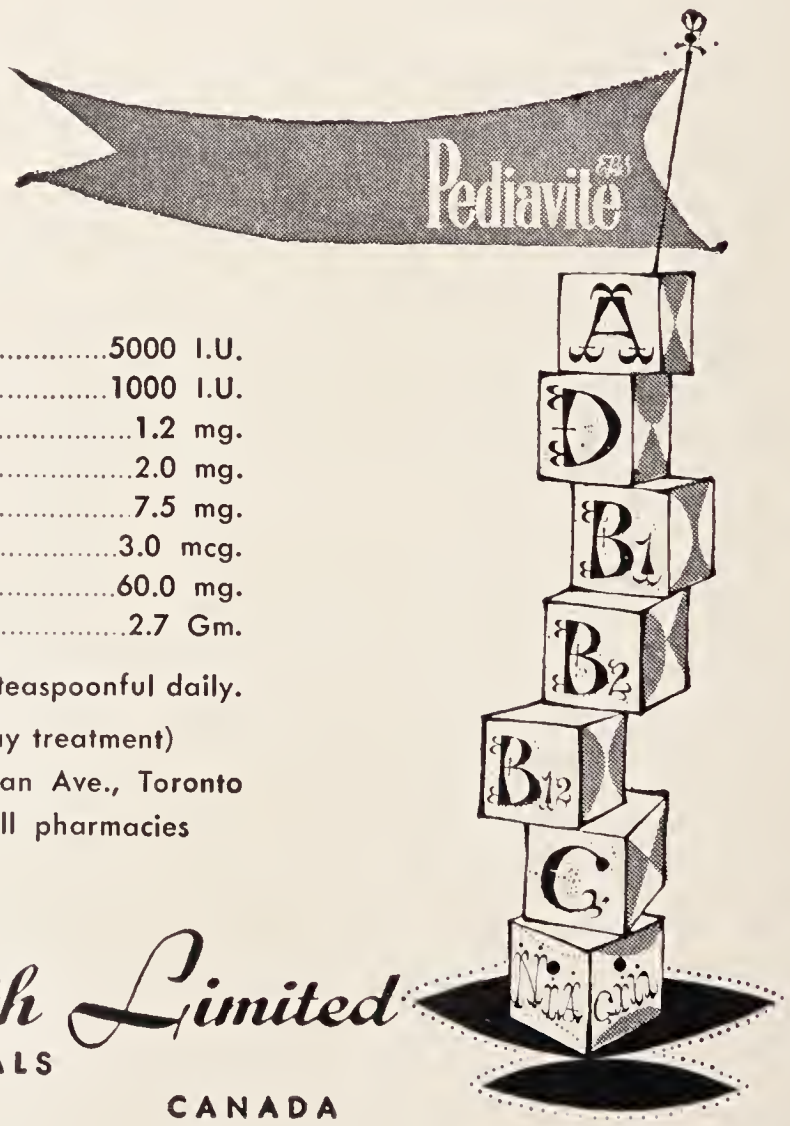
THE HOMEWOOD SANITARIUM OF GUELPH, ONTARIO, LIMITED

POTENCY ASSURED in new

Pediavite E.B.S.

FOR CHILDREN . . .

Potency of vitamins in Pediavite is assured because they are dispersed in a dry, dextrose base. In addition, Vitamins A & D have been given a special stabilization process. Pediavite is a palatable, vitamin preparation which dissolves readily in the child's formula, in a glass of milk or orange juice, or spread on cereal or other food.



Contains per level teaspoonful:

Vitamin A.	5000 I.U.
Vitamin D.	1000 I.U.
Thiamin Mononitrate	1.2 mg.
Riboflavin	2.0 mg.
Niacinamide	7.5 mg.
Vitamin B_{12}	3.0 mcg.
Ascorbic Acid	60.0 mg.
Dextrose	2.7 Gm.

75 E.B.S. 1879 • DIAMOND JUBILEE • 1954

Suggested Dosage: One level teaspoonful daily.

Package: 3 oz. jar (30 day treatment)

Write for sample to 525 Logan Ave., Toronto

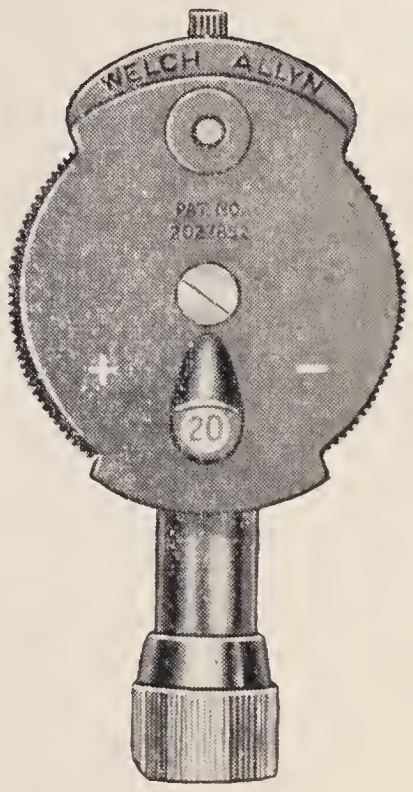
Available by ℞ . . . at all pharmacies

E. B. Shuttleworth Limited

PHARMACEUTICALS

TORONTO CANADA

. . . Accurate Diagnosis Through Instrument Quality



WHEN YOU USE WELCH ALLYN

The May type ophthalmoscope head as illustrated has a prefocused optical system which makes it unnecessary to adjust the instrument for clear focus. It also contains a patented rotable unit with standard, PIN HOLE, and SPLIT APERTURES as well as WHITE LINE GRID and RED FREE FILTER. This unit eliminates the necessity of removing the light carrier from the ophthalmoscope each time it becomes advisable to modify the projected beam of light.

A catalogue of Welch Allyn Diagnostic Instruments is available upon request.

THE JF HARTZ CO. LIMITED

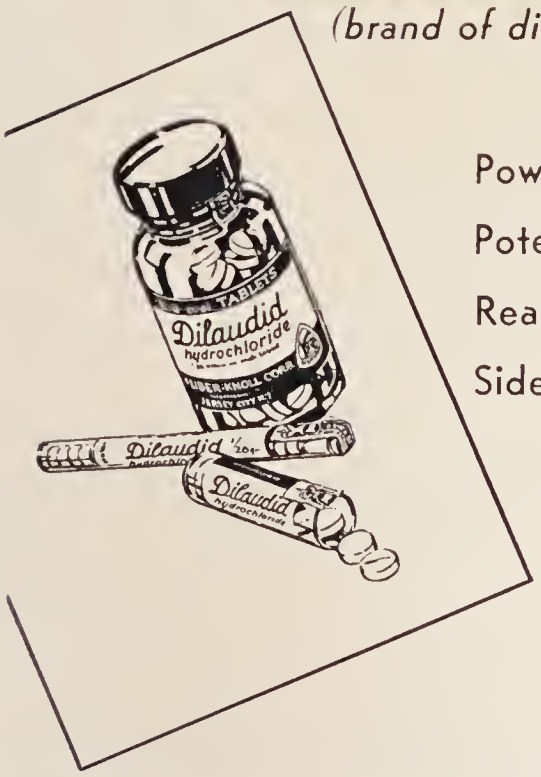
CANADIAN REPRESENTATIVE

TORONTO
MONTREAL & HALIFAX

Dilaudid hydrochloride

(brand of dihydromorphinone hydrochloride)

COUNCIL ACCEPTED



Powerful opiate analgesic - dose, 1/32 grain to 1/20 grain.

Potent cough sedative - dose, 1/128 grain to 1/64 grain.

Readily soluble, quick acting.

Side effects, such as nausea and constipation, seem less likely to occur.

An opiate, has addictive properties.

Dependable for relief of pain and cough, not administered for hypnosis.

● Dilaudid, T. M. Reg. Canada

Product of Bilhuber Knoll Corp., Orange, N. J.

MERCK & CO. Ltd. Selling Agents **MONTREAL**

from the literature...

"The value of CHLOROMYCETIN in the treatment of infections due to most bacteria, the pathogenic rickettsiae, and many of the large viruses has now been well established."[1]

in typhoid fever

"Our experience...and many others all show that chloramphenicol [CHLOROMYCETIN] has an established place in the treatment of typhoid fever."[2]

in meningitis

"At the present time chloramphenicol [CHLOROMYCETIN] is recognized as a potent antibiotic whose ease of administration and prompt diffusion into serum and spinal fluid makes it a particularly useful agent in the treatment of many forms of purulent meningitis."[3]

in bacterial endocarditis

"Within ten days [after therapy with CHLOROMYCETIN was begun] there was a dramatic improvement in the patient's clinical appearance and the sedimentation rate and temperature became normal."[4]

in rickettsial diseases

"Chloramphenicol [CHLOROMYCETIN] has been used with striking success in patients with scrub typhus, murine typhus, Rocky Mountain spotted fever, and epidemic typhus."[5]

Chloromycetin®

(Chloramphenicol, Parke-Davis)

CHLOROMYCETIN is a potent therapeutic agent and, because certain blood dyscrasias have been associated with its administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain other drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy.

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2. Dodd, K.: *J. Arkansas M. Soc.* **10**:174, 1954.
3. Hanbery, J. W.: *Neurology* **4**:301, 1954.
4. Miller, G.; Hansen, J. E., & Pollock, B. E.: *Am. Heart J.* **47**:453, 1954.
5. Keefer, C. S., in Smith, A., & Wermer, P. L.: Modern Treatment, New York, Paul B. Hoeber, Inc., 1953, p. 65.

PARKE, DAVIS & CO., LTD. WALKERVILLE, ONTARIO